Pediatrics
Clinical treatment guidelines
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Table of Contents

Acronyms

ABC : Airway, Breathing, Circulation
ABG : Arterial Blood Gases
ACE : Angiotensin Converting Enzyme
ACT : Artemisinin Combination Therapy
ACTH : Adrenocorticotrophic Hormone
ADH : Antidiuretic Hormone

AHF : Acute Heart Failure
AIDS : Acquired Immunodeficiency Syndrome
ALAT : Alanine Transaminase
ALCAPA : Aberrant Left Coronary Artery from the Pulmonary Artery
ARA : Angiotensin Receptor Antagonists
ARDS : Acute Respiratory distress Syndrome
ARF : Acute Rheumatic Fever
ASLO : Anti-Streptolysin O
AST : Aspartate AminoTransferase
AVSD : Atrio Ventricular Septal defect
AVPU : Alert, Voice, Pain, Unresponsive
BBE : Benzyl Benzoate Emulsion
BCG : Bacille Calmette -Guérin
BD, BID : Twice per day
BE : Base Excess
BP : Blood Pressure
BW : Birth Weight
CAB : Circulation Airway Breathing
CBC : Complete Blood Count
CCF : Congestive Cardiac Failure
CHD : Congenital Heart disease
CK, CPK : Creatinine (Phospho) Kinase
CKD : Chronic Kidney disease
CMV : CytoMegalo Virus
CNS : Central Nervous System
COPD : Chronic Obstructive Pulmonary disease
CPR : Cardio Pulmonary Resuscitation

CRC : Corrected Reticulocyte Count
CRP : C – Reactive Protein
CSF : Cephalo Spinal Fluid
CT : Computerized Tomography
CVD : CardioVascular disease
CVS : CardioVascular System
CXR : Chest X-Ray
DIC : disseminated Intravascular Coagulation
DKA : diabetic Keto-Acidosis
DM : diabetes Mellitus
DNA : deoxyribonucleic Acid
DVT : deep Venous Thrombosis
EBV : Epstein-Barr Virus
ECG : Electrocardiogram
EEG : Electroencephalography
ENT : Ear Nose and Throat
ESR : Erythrocyte Sedimentation Rate
FBC : Full Blood Count
GER : Gasto-Eosophageal Reflux
GFR : Glomerular Filtration Rate
GTCS : Generalized Tonic Clonic Seizures
GIT : Gastro-Intestinal Tract
GORD : Gastro-Oesophageal Reflux diseases
GXM : Group and Cross-Match
Hb : Hemoglobin
HCV : Hepatitis C Virus
HHS : Hyperosmolar Hyperglycemic State
HIE : Hypoxic Ischemic Encephalopathy

HIV : Human Immunodeficiency Virus
HR : Heart Rate
HSV : Herpes Simplex Virus
HT : Hematocrite
HTN : Hypertension
HZV : Herpes Zoster Virus
ICU : Intensive Care Unit
IE : Infective Endocarditis
IM : Intra-muscular
IR : Intrarectal
INH : Isoniazide
INR : International Normalized Ratio
ITP : Idiopathic Thrombocytopenic Purpura
IU : International Units
IV : Intravenous
JVP : Jugular Venous Pressure
KD : Kidney disease
KOH : Potassium Hydroxide
LBW : Low Birth Weight
LDH : Lactate dehydrogenase
LE : Lupus Erythematosis
LGS : Lennox-Gastaut Syndrome
LFM : Life Style Modification
LFT : Liver Function Tests
LGIB : Lower Gastro-Intestinal Bleeding
LMWH : Low Molecular Weight Heparin
LP : Lumbar Puncture
LV : Left Ventricle

MAP : Mean Arterial Pressure
MCV : Mean Cell Volume
MRI : Magnetic Resonance Imaging
NHL : Non-Hodgkin's Lymphoma
NGT : Naso Gastric Tube
NPO : Nil Per Os (Nil By Mouth)
NSAID : Non Steroidal Anti Inflammatory drugs
NVE : Native Valve Endocarditis
OD : Once per day
ORS : Oral Rehydration Salts
PA : Postero-Anterior
PaO2 : Partial Pressure Oxygen
PCP : Pneumo Cystis Pneumonia
PDA : Patent ducus Arterousus
PE : Pulmonary Embolus
PEF : Peak Expiratory Flow
PEEP : Positive End Expiratory Pressure
PO : Per Os (Take orally)
PPI : Proton Pump Inhibitor
PT : Prothrombin Time
PTT : Partial Thromboplastin Time
QID : Four times a day
PUD : Peptic Ulcer disease
RBC : Red Blood Cell
RNA : Ribonucleic Acid
RHD : Rheumatic Heart diseases
RSV : Respiratory Syncytial Virus
RR : Respiratory Rate

RV : Right Ventricle
SBP : Systolic Blood Pressure
SL : Sublingual
SLE : Systemic Lupus Erythematosis
SSSS : Staphylococcal Scaled skin Syndrome
SMEI : Severe Myoclonic Epilepsy of Infancy
T4 : Thyroxine
TB : Tuberculosis
TDS, TID : Three times per day
TORCH : Toxoplasmosis Other Rubella Cytomegalovirus Herpes
TSH : Thyroid Stimulating Hormone
UGIB : Upper Gastro-Intestinal Bleeding
ULN : Upper Limit of Normal
UTI : Urinary Tract Infection
VLBW : Very Low Birth Weight
VSD : Ventricular Septal defect
VZV : Varicella-Zona Virus
WAS : Wiskott Aldrich Syndrome
WBC : White Blood Count
WHO : World Health Organization

DIDACTION

This book is especially didacted to my parents who gave me the most precious thing in life(the life itself)

1. Respiratory Diseases

1.1. Rhinitis and Rhinopharyngitis

Definition: Rhinitis and rhinopharyngitis are very common viral

infections of the nasal or pharyngeal mucosa, which occur with seasonal variations in children under 5 years old (more frequent in cold and rainy seasons)

Causes

- Most common virus : Rhinoviruses
- Other viruses: Coronaviruses, respiratory syncytial viruses, human metapneumovirus, influenza viruses, para influenza viruses, adenoviruses, enteroviruses rarely
- Other causes include allergies (in case of recurrence), iron deficiency, passive tobacco smoke

Signs and Symptoms

- Nasal congestion
- Sore throat
- Sneezing
- Productive cough
- Fever sometimes
- Watery red eyes
- Headache

Note: Suspect allergic rhinitis in case of recurrent signs of rhinitis
with itching of nose, eyes, ears and palate.

Complications

- Otitis media
- Sinusitis (in children over 6 years old)
- Tonsillitis
- Exacerbation of asthma

Management

- No specific treatment

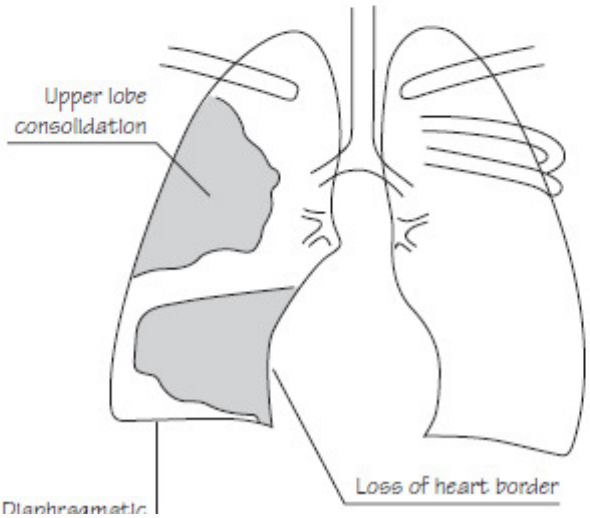
- Nasal irrigation with 0.9% Sodium chloride, 4 to 6 times/ day to clear the airway.
- In patients with fever give Paracetamol as follows:
  - 15 mg/kg/dose maximum 4 times a day (maximum dose 60mg/kg/day)
- Air humidification using nebulisation with 0.9% Sodium chloride once a day to clear the airway
- Postural drainage
- For allergic rhinitis only, give an Antihistamine (chlorpheniramine) for 3 to 5 days as follows:
  - From 2 to 5 years: 1 mg, to be repeated 4 to 6 times without exceeding 6 mg/day
  - From 6 to 12 years: 2 mg, to be repeated 4 to 6 times without exceeding 12mg/day
  - Avoiding the allergen

Recommendation

- Antibiotics are not indicated in viral rhinitis and rhinopharyngitis except in cases of evident super-infection

1.2. Pneumonia

CXR of right middle and upper lobe pneumonia



Definition: Pneumonia is an inflammation of the parenchyma of the lungs classified according to the infecting organism.

Causes

- Bacterial: Streptococcus pneumonia is the most common at all ages followed by Chlamydia pneumonia and Mycoplasma pneumonia (over 5 year old age), Chlamydia trachomatis
(infant) Staphylococcus aureus, Haemophilus influenza (in case of no vaccination), Pseudomonas aeruginosa (In immunocompromised patients), Klebsiella pneumonia
- Viral: Respiratory Synctitial Virus, Adenovirus, Influenzae A and B, Parainfluenzae types 1 and 3, Metapneumovirus
- Fungal: Cryptococcus neoformans, Aspergillus spp
- Mycobacterial: Mycobacterium tuberculosis, Mycobacterium avium, Mycobacterium intracellulare,
- Parasites: Pneumocystis jiroveci

Signs and Symptoms

- Fever
- Tachypnea
- Respiratory distress (inter-costal, sub-costal recession)
- Nasal flaring
- Use of accessory muscles
- Cyanosis and respiratory fatigue (in severe case especially for infant)
- Crackles and wheezing in auscultation
- bronchial breathing

Findings suggestive of viral and bacterial pneumonia

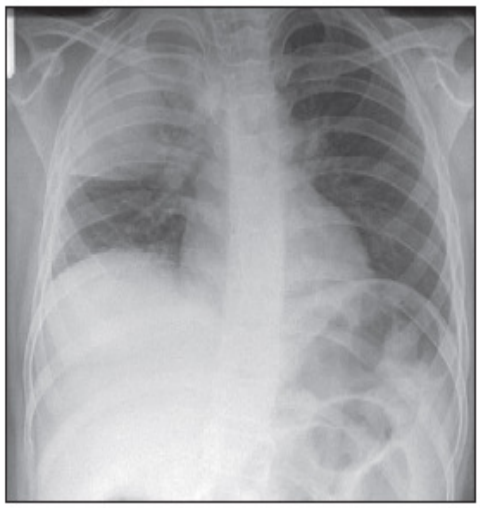
Findings	Viral pneumonia	Bacterial pneumonia
Initial signs	Upper respiratory tract infection	Upper respiratory tract infection (in case of super-infection)
Fever	Low	High
Pulmonary sign	Tachypnea Bronchial, crackles	Tachypnea Crackles
Clinical signs		
WBC	<20000 Lymphocytes predominance	15000-40000 Granulocytes predominance
Inflammatory test (CRP and ESR)	Low	High
Chest X-Ray	Perihilar changes Diffuse findings on chest exam are common Often peribronchial thickening	Alveolar pneumonia Bronchopneumonia usually bilateral Lobar pneumonia Lung abscess

Note: It is often not possible to distinguish viral pneumonia from disease caused by bacterial pathogens.

Type	Signs	Symptoms
Very severe pneumonia	Cyanosis Inability to drink/breastfeed AVPU = V, P or U Grunting	History of cough or difficulty breathing Fever Abdominal/chest pain (sometimes)
Severe pneumonia	Lower chest indrawing Nasal flaring grunting	
Non severe Pneumonia	Fast breathing presence or absence of crackles	

Complications
- Empyema
- Pleural effusion
- Pneumothorax
- Sepsis/ Meningitis / Arthritis

Investigations
- FBC
- Chest x-ray



Consolidation of the right upper lobe.

Infectious Pneumonia

Frontal radiograph shows focal consolidation ⇨ within the superior segment of the left lower lobe without associated volume loss, consistent with pneumonia in this toddler with a cough and fever.

- Blood culture
- HIV test

Management

Criteria for hospitalization

• Community acquired pneumonia can be treated at home

• Identify indicators of severity in children who need admission, as pneumonia can be fatal. The following indicators can be used as a guide for admission:

• Children aged 3 months and below, whatever the severity of pneumonia.

• Fever (more than 38.5 ^{o}C), refusal to feed and vomiting
• Fast breathing with or without cyanosis
• Associated systemic manifestation
• Failure of previous antibiotic therapy
• Recurrent pneumonia
• Severe underlying disorder, e.g. Immunodeficiency
- Severe respiratory distress
• Supplemental oxygen
• dehydration
• Vomiting
• No response to appropriate oral antibiotic therapy

Management summary of pneumonia

Type	Management	Comments
Very severe pneumonia	- Hospitalization - Oxygen - Correct shock, hypoglycaemia and dehydration - Fluid maintenance - *Ampicillin* 200mg/kg Q6hr ***or*** *Benzyl penicillin* 50,000 units/kg IM/IV Q6hr **Plus** - *Gentamycine* IV 7.5mg/kg IV over 3-5 minutes Q24hr ***Or*** - *Cefotaxime* 50mg/kg/dose Q8hr **(second line)**	Duration 10 days Switch to oral treatment with amoxicillin if improvement in clinical symptoms
Severe pneumonia	- Hospitalization - Oxygen - Correct hypoglycaemia and dehydration - Fluid maintenance - *Ampicillin* 100mg /kg/day (33 mg/ kg/dose Q8h)	Duration 7 days
Non severe Pneumonia	- *Amoxicillin* 50-mg/kg/dose Q12hr	Duration 5 days

Note: If pneumonia due to staphylococcus is suspected give *Cloxacillin* 100mg/kg/day for 7 days in 3doses and *Gentamycine* Use vancomycin as second line therapy if no response.

Recommendations

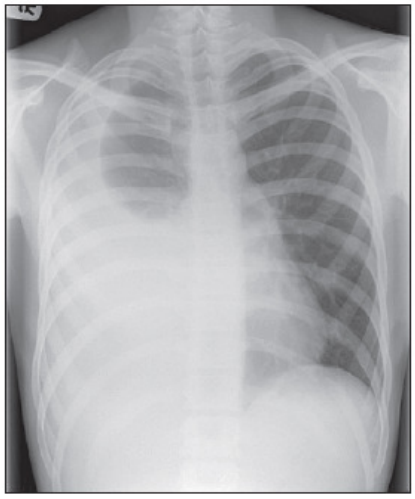
- Recurrent/persistent pneumonia

In case of persistent pneumonia (abnormal X-ray more than30 days after treatment) the patient should be referred for
investigations (CT Scan, bronchoscopy) to exclude:

- Foreign body
- Congenital malformation (adenomatosis)
- Immotile cilia syndrome

- Likewise, in case of recurrent pneumonia, an underlying cause should be suspected and the child referred for further investigations.

• Pleural effusion



Right-sided empyema.

- In case of pleural effusion, think of Staphylococcus aureus,
streptococcus pneumonia, mycoplasma pneumonia, tuberculosis

• Exclude Tuberculosis
• Ultrasound to measure the volume of liquid and aspiration for culture
• drainage of fluid if important and respiratory distress

1.3. Wheezing child/Asthma and Bronchiolitis

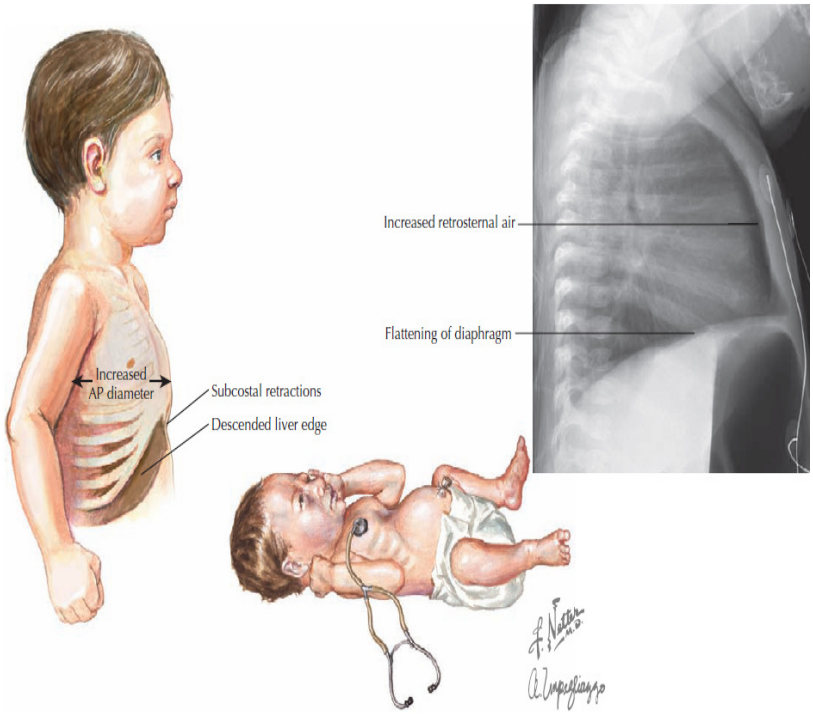
Wheezing child

Definition: A wheeze is a musical and continuous sound that originates from oscillations in narrowed airways. Wheezing is heard mostly in expiration as a result of critical airway obstruction.

Causes/ Risk factors

- Bronchiolitis
- Asthma
- Esophageal foreign bodies
- Aspiration syndrome (gastro-esophageal reflux diseases)

1.3.1. Acute Bronchiolitis



Definition: Bronchiolitis is an inflammation of the bronchiole tubes
due to viral organism resulting in wheezing. In children under 2 years old, it may lead to fatal respiratory distress. Occurs with seasonal
variations and has epidemic potential.

Causes

- Acute bronchiolitis is predominantly a viral disease
- Respiratory Syncytial Virus is the most common (>50% cases)

- Other agents: parainfluenza, adenovirus, Mycoplasma, and occasionally other viruses especially Human metapneuo virus

Clinical signs

- dyspnea with cough (both day and night)
- distension of the thorax
- Low-grade fever
- Prolonged expiration with diffuse wheeze on pulmonary auscultation
- Occasionally fine, diffuse, bilateral late inspiratory crepitations
- Signs of serious illness include tachypnea, central cyanosis (tongue and gingiva), Nasal flaring, Chest indrawing, periods of apnoea, altered level of consciousness, difficulty drinking or breastfeeding, and silence on auscultation (corresponding to an intense bronchospasm)

Complications

- Bacterial secondary infection
- Atelectasis
- Apnoea especially in neonatal and infant period

Investigations

- FBC
- CRP (Less contributory as viral infection)
- Chest X-ray: show hyperinflated lungs with patchy atelectasis

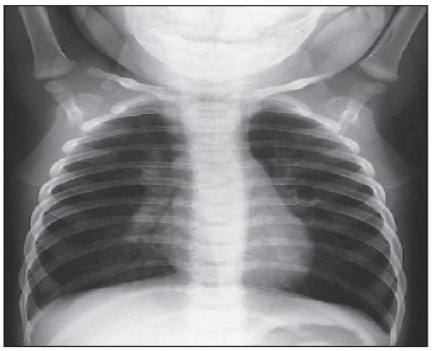


Figure 16.6 In acute bronchiolitis, the chest X-ray shows hyperinflation of the lungs with flattening of the diaphragm, horizontal ribs and increased hilar bronchial markings. However, chest X-ray is rarely helpful in bronchiolitis.

- Viral testing (usually rapid immunofluorescence, polymerase chain reaction, or viral culture) is helpful if the diagnosis is uncertain or for epidemiologic purposes

Management

Guidelines for Hospital Admission in Viral Bronchiolitis		
	Home Management	Hospital management
Age < than 3 months	No	Yes
Toxic – looking	No	Yes
Chest recession	Mild	Moderate/Severe
Central cyanosis	No	Yes
Wheeze	Yes	Yes
Crepitations on auscultation	Yes	Yes
Feeding	Well	Difficult
Apnoea	No	Yes
Oxygen saturation	> 95%	< 93 %
High risk group	No	Yes

- Treatment is symptomatic
- Hospitalize children if signs of serious illness
- Administer high humidified oxygen at 8L/min in 30 to 40 % oxygen
- Attention to pulmonary toilet including suctioning,
percussion and postural drainage
- IV fluid > maintenance
- Tube feeding when the child is in improved respiratory distress state
- In case of respiratory failure, use non-invasive nasal CPAP or mechanical ventilation

Recommendations

- *Antibiotics* are recommended for all infants with
- Recurrent apnoea and circulatory impairment.

- Possibility of septicaemia.
- Acute clinical deterioration.
- High white cell count.
- Progressive infiltrative changes on chest radiograph.
- Give oral or parenteral antibiotics for 5 days based on severity and/or condition of the patient as follow:

• Amoxicillin 25mg per dose/kg/day Q12hr PO

Or

• Ampicillin injections IM: 100 mg/kg/day in 3 divided doses or

- Alternative treatment:

• Erythromycin 30 -50 mg per dose/kg/day x3/day/7-10days

Note: Evidence on Treatment of bronchospasms does not support routine use of bronchodilators, steroids or antibiotics.

If bronchodilators are to be used, closely monitor effect as it might worsen respiratory distress

1.3.2. Asthma

Definition: Asthma is a chronic inflammatory condition of the lung airways resulting in episodic airflow obstruction.

Causes

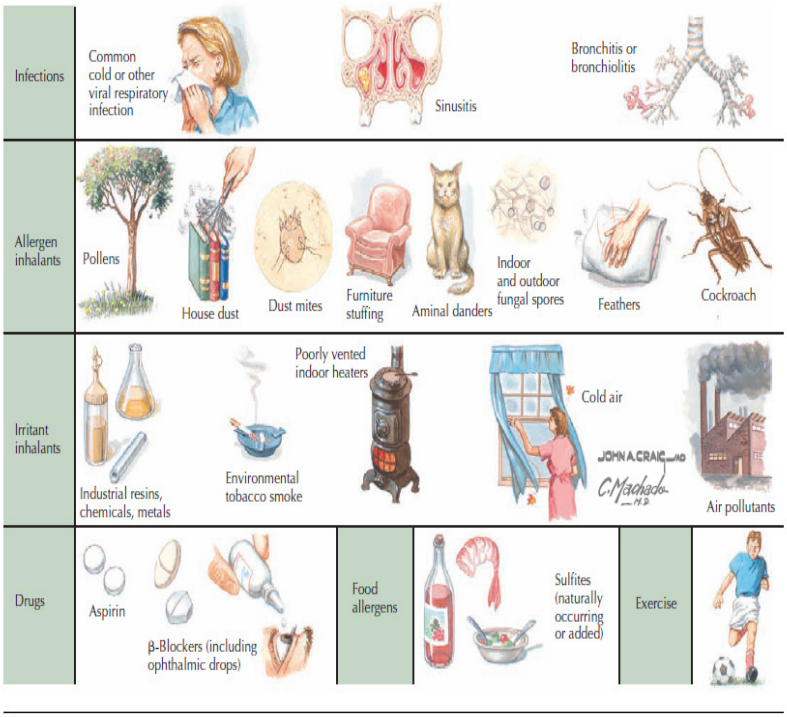


Figure 38-3 *Asthma triggers.*

- Unknown but the following factors have been identified:

•Allergens (e.g., house dust, perfumes, food, animal airs, mites)

- Medicine (e.g., propranolol and aspirin)
- Environmental (e.g., change of weather, polluants), Infections (viral or bacterial)
- Emotions
- Family history (genetic factors)

- Gastro-esophageal reflux

Signs and Symptoms

Asthma

Inspection of chest shape is an important part

boy with poorly controlled asthma has an inc

- The depressions at the base of the thorax associated with the muscular insertion of the diaphragm are called Harrison sulci, and are associated with chronic obstructive airways disease such as asthma during childhood.

Breathlessness

- Wheezing/ prolonged expiratory
- Cough (chronic nocturnal cough)

- Exercise induced cough
- Chest tightness
- Sputum production

Severity of Asthma Exacerbation

Parameter	Mild	Moderate	Severe	Respiratory arrest imminent
Breathless	Walking Can lie down	Talking Infant - softer, shorter cry; difficulty feeding Prefers sitting	At rest Infant stops feeding Hunched forward	
Talks in	Sentences	Phrases	Words	
Alertness	May be agitated	Usually agitated	Usually agitated	Drowsy or confused
Respiratory rate	Increased	Increased	Greatly increased	
Normal rates of breathing in awake children < 2 months: < 60/min 2-12 months: < 50/min 1-5 years: < 40/min 6-8 years: < 30/min				
Accessory muscles and suprasternal retractions	Usually not	Usually	Usually	Paradoxical thoraco-abdominal movement
Wheeze	Moderate, often only and expiratory	Loud	Usually loud	Absence of wheeze

Severity of Asthma Exacerbation (cont.)

Parameter	Mild	Moderate	Severe	Respiratory arrest imminent
Pulse/min.	<100	100 - 200	>120	Bradycardia
Guide to limits of normal pulse rate in children				
Infants	2-12 months	< 160/min		
Preschool	1-2 years	< 120/min		
School age	2-8 years	< 110/min		
Pulsus paradoxus	Absent < 10 mm Hg	May be present 10 - 25 mm Hg	Often present > 25 mm Hg (adult) 20 - 40 mm Hg (children)	Absence suggests respiratory muscle fatigue
PEF after initial bronchodilator % predicted or % personal best	Over 80%	Approx. 60-80%	< 60% predicted or personal best or response lasts < 2 hrs	
PaO2 (on air)† and/or paCO2†	Normal < 45 mm Hg *Test not usually necessary!*	>60 mm Hg < 45 mm Hg	< 60 mm Hg > 45 mm Hg *Possible cyanosis and respiratory failure!*	
SaO2% (on air)†	>95%	91 - 95%	<90%	
Hypercapnia (hyperventilation) develops more readily in young children than in adults and adolescents				
*Note: *The presence of several parameters, but no necessarily all, indicates the general classification of the exacerbation*				

Levels of Asthma Control (GINA 2006)			
Characteristics	*Controlled* All of the following:	*Partly Controlled* *Any* measure present in *any* week:	*Uncontrolled*
Daytime symptoms	None	> 2 per week	≥ 3 features of partly controlled asthma present in any week
Limitation of activities	None	Any	
Nocturnal symptoms or Awakenings	None	Any	
Need for Reliever	None	> 2 per week	
Lung function test	Normal	< 80% predicted or personal best	
Exacerbations	None	≥ 1 per year	One in *any* week

Signs and Symptoms

Note: Asthma can often be diagnosed on the basis of a patient's symptoms and medical history.

Presence of any of these signs and symptoms should increase the suspicion of asthma:

- Wheezing high-pitched whistling sounds when breathing out-especially in children.
- History of any of the following:

• Cough, worse particularly at night
• Recurrent wheeze
• Recurrent difficult breathing
• Recurrent chest tightness
• Symptoms occur or worsen at night, awakening the patient
• Symptoms occur or worsen in a seasonal pattern

• The patient also has eczema, hay fever, or a family history of asthma or atopic diseases

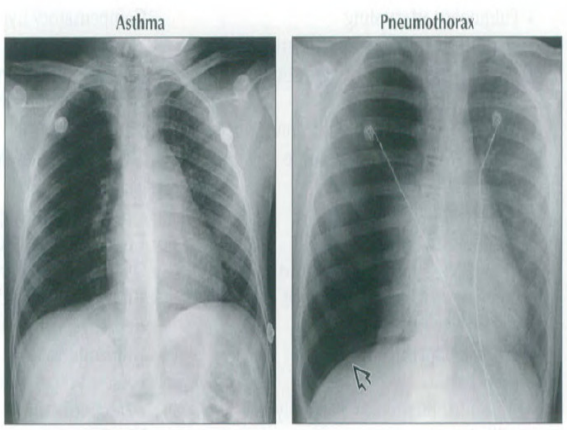
- Symptoms occur or worsen in the presence of:
  - Animals with fur
  - Aerosol chemicals
  - Changes in temperature
  - domestic dust mites
  - drugs (aspirin, beta blockers)
  - Exercise
  - Pollen
  - Respiratory (viral) infections
  - Smoke
  - Strong emotional expression
- Symptoms respond to anti-asthma therapy
- Patients colds “go to the chest” or take more than 10 days to clear up

Complications

- Uncontrolled/poorly controlled asthma can lead to severe lung damage
- Severe asthma exacerbation can cause respiratory failure and death

Investigations

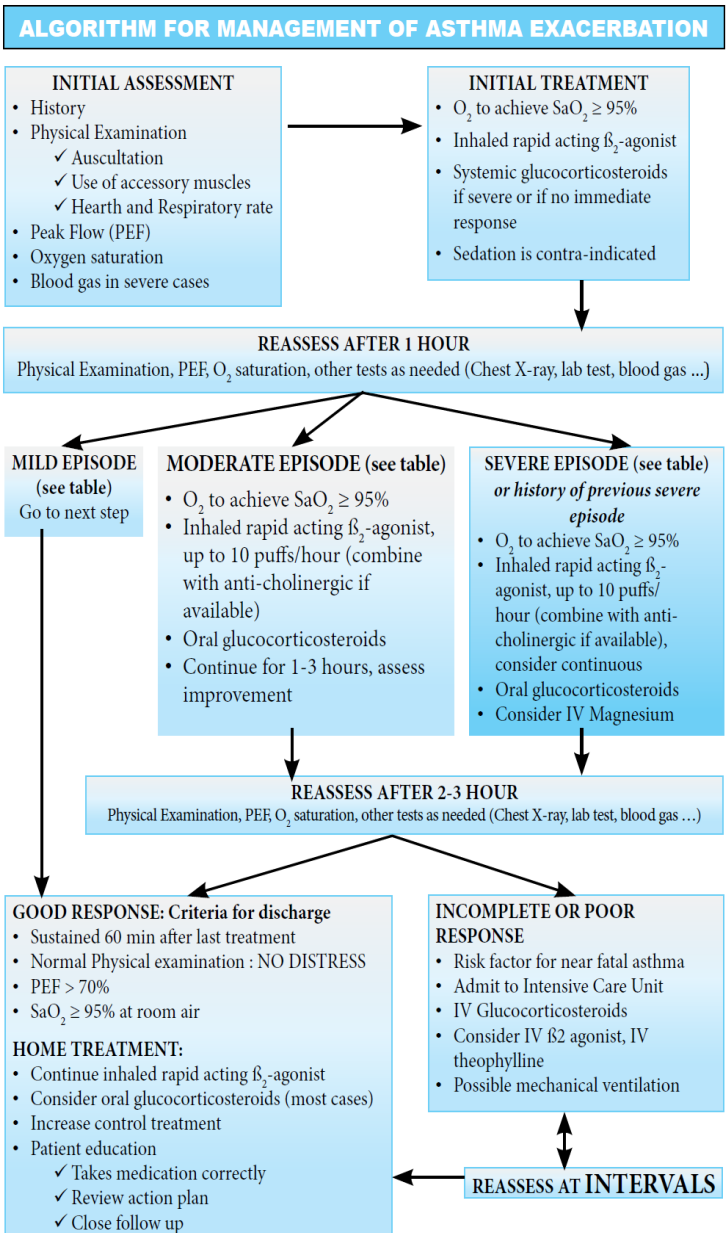
- Lung function to confirm diagnosis and assess severity
- Peak expiratory flow rate can help diagnosis and follow up
- Additional diagnostic tests
  - Allergy testing (where applicable)
  - Chest X-ray (for differential diagnosis)



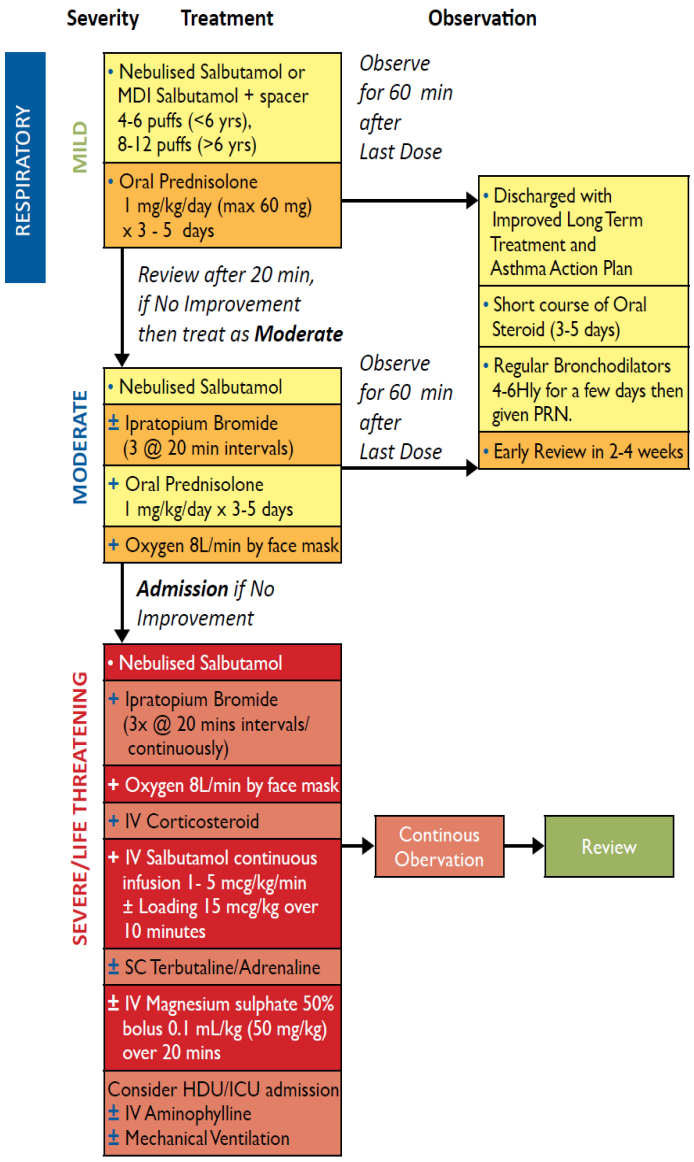
(Left) Frontal radiograph shows diffuse relative hyperlucency and hyperexpansion of the right lung compared with the left in this 8 year old with known asthma. The appearance resolved after nebulizer treatment. (Right) Frontal radiograph in this 13 year old shows a large, right, spontaneous pneumothorax resulting in a hyperlucent right hemithorax. There is mild depression of the right hemidiaphragm, indicating a tension component.

- FBC for exclusion of super-infection

Management


ALGORITHM FOR MANAGEMENT OF ASTHMA EXACERBATION
INITIAL ASSESSMENT
• History
• Physical Examination
✓ Auscultation
✓ Use of accessory muscles
✓ Hearth and Respiratory rate
• Peak Flow (PEF)
• Oxygen saturation
• Blood gas in severe cases
INITIAL TREATMENT
• O2 to achieve SaO2 ≥ 95%
• Inhaled rapid acting ß2-agonist
• Systemic glucocorticosteroids if severe or if no immediate response
• Sedation is contra-indicated
REASSESS AFTER 1 HOUR
Physical Examination, PEF, O2 saturation, other tests as needed (Chest X-ray, lab test, blood gas ...)
MILD EPISODE (see table)
Go to next step
MODERATE EPISODE (see table)
• O2 to achieve SaO2 ≥ 95%
• Inhaled rapid acting ß2-agonist, up to 10 puffs/hour (combine with anti-cholinergic if available)
• Oral glucocorticosteroids
• Continue for 1-3 hours, assess improvement
SEVERE EPISODE (see table)
or history of previous severe episode
• O2 to achieve SaO2 ≥ 95%
• Inhaled rapid acting ß2-agonist, up to 10 puffs/hour (combine with anti-cholinergic if available), consider continuous
• Oral glucocorticosteroids
• Consider IV Magnesium
REASSESS AFTER 2-3 HOUR
Physical Examination, PEF, O2 saturation, other tests as needed (Chest X-ray, lab test, blood gas ...)
GOOD RESPONSE: Criteria for discharge
• Sustained 60 min after last treatment
• Normal Physical examination : NO DISTRESS
• PEF > 70%
• SaO2 ≥ 95% at room air
HOME TREATMENT:
• Continue inhaled rapid acting ß2-agonist
• Consider oral glucocorticosteroids (most cases)
• Increase control treatment
• Patient education
✓ Takes medication correctly
✓ Review action plan
✓ Close follow up
INCOMPLETE OR POOR RESPONSE
• Risk factor for near fatal asthma
• Admit to Intensive Care Unit
• IV Glucocorticosteroids
• Consider IV ß2 agonist, IV theophylline
• Possible mechanical ventilation
REASSESS AT INTERVALS

Management of Acute Exarcerbation of Bronchial Asthma in Children



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Stepwise approach for maintenance treatment

Level of control	Treatment action
Controlled	Maintain and find lowest controlling step
Partially controlled	Consider stepping up to gain control
Uncontrolled	Step up until controlled
Exacerbation	Treat exacerbation

Management Based On Control				
← Reduce			Increase →	
STEP 1 Intermittent	**STEP 2** Mild Persistent	**STEP 3** Moderate Persistent	**STEP 4** Severe Persistent	**STEP 5** Severe Persistent
As needed rapid acting β_2-agonist	As needed rapid acting β_2-agonist			
Controller Options	*Select one*	*Select One*	*Add one / more*	*Add one / both*
	Low dose inhaled steroids	Low dose ICS + long acting β_2-agonist	Medium / High dose ICS + long acting β_2-agonist	Oral Glucocorticoids lowest dose
	Leukotriene modifier	Medium / High dose ICS	Leukotriene modifier	Anti-IgE
		Low dose ICS + Leukotriene modifier	SR Theophylline	
		Low dose ICS + SR Theophylline		

Footnote: ICS, Inhaled Corticosteroids; SR, Sustained Release.

Drug Dosages for Medications used in Chronic Asthma		
Drug	**Formulation**	**Dosage**
Relieving Drugs		
β_2-agonists		
• Salbutamol	Oral Metered dose inhaler Dry powder inhaler	0.15 mg/kg/dose TDS-QID/PRN 100-200 mcg/dose QID/PRN 100-200 mcg/dose QID/PRN
• Terbutaline	Oral	0.075 mg/kg/dose TDS-QID/PRN 250-500 mcg/dose QID/PRN 500-1000 mcg/dose QID/PRN (maximum 4000 mcg/daily)
• Fenoterol	Metered dose inhaler	200 mcg/dose QID/PRN
Ipratropium Bromide	Metered dose inhaler	40-60mcg /dose TDS/QID/PRN
Preventive Drugs		
Corticosteroids		
• Prednisolone	Oral	1-2 mg/kg/day in divided doses
• Beclomethasone Diproprionate • Budesonide	Metered dose inhaler Dry powder inhaler	<400 mcg/day : low dose 400-800 mcg/day : Moderate 800-1200 mcg/day: High
• Fluticasone Propionate	Metered dose inhaler Dry powder inhaler	<200 mcg/day : Low 200-400 mcg/day : Moderate 400-600 mcg/day : High
• Ciclesonide	Metered dose inhaler	160 microgram daily 320 microgram daily
Sodium Cromoglycate	Dry powder inhaler Metered dose inhaler	20mg QID 1-2mg QID or 5-10mg BID-QID
Theophylline	Oral Syrup Slow Release	5 mg/kg/dose TDS/QID 10 mg/kg/dose BD
Long acting β_2-agonists		
• Salmeterol	Metered dose inhaler Dry powder inhaler	50-100 mcg/dose BD 50-100 mcg/dose BD
Combination		
Salmeterol / Fluticasone	Metered dose inhaler Dry powder inhaler	25/50mcg, 25/125mcg, 25/250mcg 50/100mcg,50/250mcg, 50/500mcg
Budesonide /Formoterol	Dry powder inhaler	160/4.5mcg, 80/4.5mcg
Antileukotrienes (Leukotriene modifier)		
Montelukast	Oral	4 mg granules 5mg/tablet on night chewable 10mg/tablet ON

Footnotes on Management of Acute Exacerbation of Asthma:

1. Monitor pulse, colour, PEFR, ABG and O2 Saturation. Close monitoring for at least 4 hours.
2. Hydration - give maintenance fluids.
3. Role of Aminophylline debated due to its potential toxicity. To be used with caution, in a controlled environment like ICU.
4. IV Magnesium Sulphate : Consider as an adjunct treatment in severe
exacerbations unresponsive to the initial treatment. It is safe and beneficial in severe acute asthma.
5. Avoid Chest physiotherapy as it may increase patient discomfort.
6. Antibiotics indicated only if bacterial infection suspected.
7. Avoid sedatives and mucolytics.
8. Efficacy of prednisolone in the first year of life is poor.
9. On discharge, patients must be provided with an Action Plan to assist parents or patients to prevent/terminate asthma attacks.
The plan must include:
a. How to recognize worsening asthma.
b. How to treat worsening asthma.
c. How and when to seek medical attention.
• Salbutamol MDI vs nebulizer
< 6 year old: 6 x 100 mcg puff = 2.5 mg Salbutamol nebules.

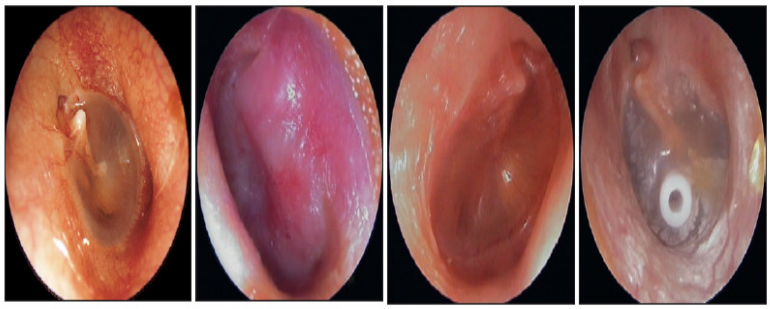
- 6 year old: 12 x 100 mcg puff = 5.0 mg Salbutamol nebules.

Drug Dosages for Medications used in Acute Asthma		
Drug	**Formulation**	**Dosage**
β_2-agonists		
• Salbutamol	Nebuliser solution 5 mg/ml or 2.5 mg/ml nebule Intravenous	0.15 mg/kg/dose (max 5 mg) or < 2 years old : 2.5 mg/dose > 2 years old : 5.0 mg/dose Continuous : 500 mcg/kg/hr Bolus: 5-10 mcg/kg over 10 min Infusion: Start 0.5-1.0 mcg/kg/min, increase by 1.0 mcg/kg/min every 15 min to a max of 20 mcg/kg/min
• Terbutaline	Nebuliser solution 10 mg/ml, 2.5 mg/ml or 5 mg/ml respule Parenteral	0.2-0.3 mg/kg/dose, or < 20 kg: 2.5 mg/dose > 20 kg: 5.0 mg/dose 5-10 mcg/kg/dose
• Fenoterol	Nebuliser solution	0.25-1.5 mg/dose
Corticosteroids		
• Prednisolone	Oral	1-2 mg/kg/day in divided doses (for 3-7 days)
• Hydrocortisone	Intravenous	4-5 mg/kg/dose 6 hourly
• Methylprednisolone	Intravenous	1-2 mg/kg/dose 6-12 hourly
Other agents		
Ipratropium bromide	Nebuliser solution (250 mcg/ml)	< 5 years old : 250 mcg 4-6 hourly > 5 years old : 500 mcg 4-6 hourly
Aminophylline	Intravenous	6 mg/kg slow bolus (if not previously on theophylline) followed by infusion 0.5-1.0 mg/kg/hr
Montelukast	Oral	4 mg granules 5mg/tablet on night chewable 10mg/tablet ON

-

1.4. Ear Nose and Throat Conditions

1.4.1. Acute Otitis Media



(a) (b) (c) (d)

Figure 16.2 Appearance of the eardrum. (**a**) Normal. (**b**) Acute otitis media. (**c**) Otitis media with effusion. (**d**) Grommet. (Courtesy of Mr N Shah & Mr N Tolley.)

Definition: It is the inflammation of the middle ear cavities.

Causes

- Viral
- Bacterial (Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis etc.)
- Predisposing factors include poor living conditions, adenoids, sinusitis, allergic rhinitis, tonsillitis, asthma etc.

Signs and Symptoms

- Fever
- Retroauricular pain
- Crying with ear scrubbing
- Gastro intestinal signs
- Otalgia
- Cervical lymphadenopathy
- Otorrhea (if tympanic membrane perforated)

- Impaired hearing
- Redness of eardrum
- Sometimes bulging of the eardrum

Complications

- Secretory otitis media (ear glue)
- Chronic otitis media with perforation
- Acute mastoiditis sometimes with periosteal abscess
- Intracranial (meningitis, brain abscess, subdural abscess, etc)
- Facial paralysis
- Labyrinthitis

Investigations

- Clinical including otoscopy
- FBC and CRP if signs of sepsis

Management

- General measures: elimination of risk factors

Pharmacological

- Treatment of first choice

• Amoxicillin, Po 30mg/kg/dose P.O. Q8h for 7-10 days
• When associated with rhinitis add Xylometazoline (Otrivine) 0.5% nose drops or simple argyrol drops 1% , 0.05%
•Paracetamol 10-15mg/kg/dose Q6hr if high fever or pain

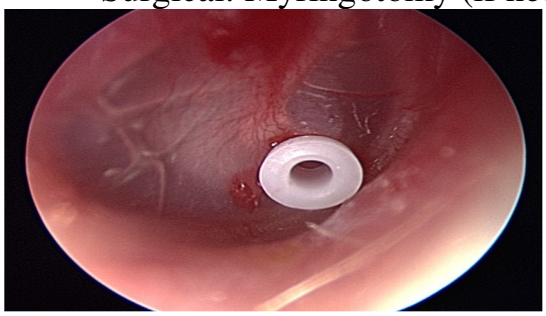
- Alternative treatment

• Amoxi-clav (Augmentin) 50mg/kg/day P.O, Q8h for 7 -10 days;

Or cefadroxyl : 25mg/kg/dose Q12h for 7 days

• cefuroxime (Zinat): 15mg/kg /dose Q12h for 7 days
• Azithromycine 10mg/kg/dose Q24h for 3 days
• Erythromycine 20 mg/kg/dose Q8h for 10 days

- Surgical: Myringotomy (if necessary)



Note tube sitting in antero-inferior quadrant of tympanic membrane to avoid damage to the structures of the middle ear.

nendation

- Avoid getting in the inside of the wet ear

1.4.2. Chronic Suppurative Otitis Media

Definition: It is a chronic inflammation of the middle ear with
recurrent ear discharges or otorrhoea through a tympanic perforation for more than 2 weeks.

Predisposing risk factors

- Inadequate management of otitis media
- Frequent upper respiratory tract infections
- Anatomic factor: short eustachian tube
- Poor living conditions, poor housing, hygiene and nutrition analphabetism
- Immunosupression (e.g.: HIV infection)

Causes
- Tuberculosis
- P. aeruginosa
- S.pneumoniae
- Staphyllococcus aureus
- H. Influenza

Signs and Symptoms
- Recurrent pus ear discharge
- Large perforation of the eardrum on examination
- Progressive hypoacousia with impaired hearing
- Buzzing (acouphene)
- History of recurrent otitis media
- Loss of transparency of tympanic membrane

Complications
- Subperiosteal abscesses
- Facial nerve paralysis
- Lateral sinus thrombophlebitis
- Suppurative labyrinthitis
- Brain abscess
- Meningitis
- Mastoiditis
- Extradural and subdural Empyema
- Otitic hydrocephalus
- Hearing impairment
- deafness

Investigations
- Bacterial Cultures
- Search for predisposing factors

- Audiogram
- CT-scan

Management

Non pharmacological management

• dry mopping
• Aural toilet by medicines' droppers (with Hydrogen peroxide or polyvidone iodine saline solutions)
• Avoid getting the inside of the ear wet. e.g.: bathing and swimming

Pharmacological management

• Topical quinolones (ciprofloxacin ear drops Q12h for 7 days)
• Systemic treatment: ceftazidime IV or IM 50mg/kg/dose Q8h (max:6gr/day) for 7 days Surgical
• In case of mastoiditis: Mastoidectomy

Recommendations

- Proper management of acute otitis media
- Avoid getting the inside of the ear wet. e.g: bathing and swimming
- Refer to the tertiary health facility for further management

1.4.3. tonsillitis

Fig. 53 Tonsillitis. The tonsils are enlarged, hyperaemic, superficially ulcerated and meet in the midline. In association with adenoidal hypertrophy, this may result in significant upper airways obstruction. Gagging during examination may draw the tonsils forward leading to artefactual hypertrophy.

Acute Tonsillitis
Note the enlarged tonsils bilaterally (L>R) with purulent exudates

Definition: It is an inflammation of the tonsils
Causes
- Bacterial infection (Group A β-hemolytic streptococcal, staphylococcal)
- Viral infection (Rhinoviruses, influenza)
- Fungal infection

Signs and Symptoms
- difficult and painful swallowing (dysphagia)
- Refusal of breastfeeding
- Fever, chills
- Headache
- Vomiting
- Sore throat - lasts longer than 48 hours and may be severe
- Enlarged and tender submandibular lymph nodes
- Swollen red tonsils with white spots

Complications
- Rheumatic heart disease
- Acute glomerulonephritis
- Middle ear infections
- Peritonsillar abscess (quinsy)
- Abscess of the pharynx
- Sinusitis
- Septicaemia
- Bronchitis or pneumonia
- Airway obstruction

Investigations
- Swab for laboratory analysis

- Complete blood count if signs of sepsis
- Streptococcal screen

Management

Medical treatment

• Ensure enough fluids to avoid dehydration
• Amoxicillin 15-30 mg/kg/dose Q8h for 10 days

Or

• Penicillin V tabs: 15mg/kg/dose Q12h for 10days

Or

• Erythromycine 15-20mg/kg/dose Q8h for 10 days Or Azithromycine 10mg/kg/dose Q24h for 3 days In case of allergy to penicillins use
• If fever or pain, give Ibuprofen: 2-3mg/kg/dose Q8h

Or Paracetamol 10-15mg/kg Q6h, max 60mg/kg/day

If no response with the first choice

• Amoxi-clav (Augmentin) 15-20mg/kg/dose P.O, Q8h 7 -10 days;

Or

• cefuroxime (Zinat): 15mg/kg /dose Q8h for 7 days

Surgical treatment

• Tonsillectomy indicated in:

→Chronic repetitive tonsillitis

→ Obstructive tonsils

Recommendations

- Systematically give Antibiotherapy to children > 3 years in order to prevent rheumatic heart disease
- For chronic and obstructive tonsillitis refer to the ENT specialist

1.4.4. Acute Mastoiditis

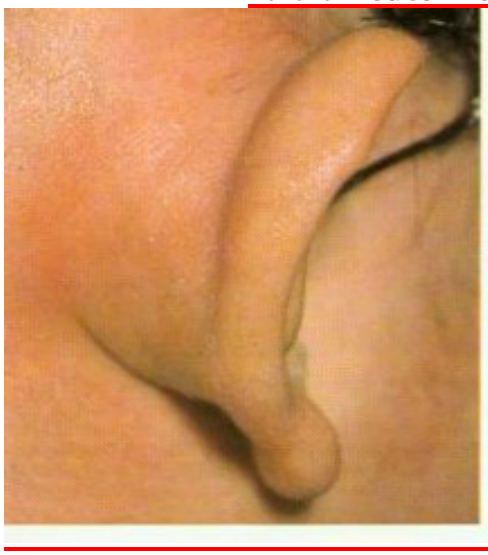


Fig. 55 Mastoiditis. Infection of the mastoid air cells, usually from otitis media, has produced overlying erythema and oedema, resulting in the forward and downward displacement of the external ear. Osteitis should be managed surgically.

Definition: Acute mastoiditis is sudden onset bacterial infections of the mastoid bone

Cause

- Spread of pathogens causing acute otitis media to the mastoid bone

Signs and Symptoms

- Fever
- Pain, tenderness, discomfort and swelling behind the ear
- In some instances, the ear on the affected side seems pushed out and quite prominent: this is caused by a high concentration of pus in the mastoid
- Sometimes associated suppurative otitis media

- Tympanic membrane is usually perforated with otorrhoea
- Occasionally, pus breaks through the mastoid tip and forms an abscess in the neck (Bezold's abscess)
- Headache
- Hearing loss

Complications

- Facial paralysis
- Brain abscess
- Meningitis
- Neck abscess
- Extradural abscess
- Septicemia
- Subdural abscess

Investigations

- X-Ray of the mastoid bone
- In selected cases
  - CT-scan of the middle ear
  - Culture of the pus from the mastoid bone
  - Hemoculture
  - LP if signs of meningitis

Management

Pharmacological

- Treatment of first choice

→ cephalosporine 3rd generation:

- cefotaxime IV 30-50 mg/kg/dose Q8h for 7-10 days

Or

- ceftriaxone IV 100mg/kg/dose Q24h for 7-10 days

→ If 3rd generation cephalosporine not available

- Ampicillin IV 50mg/kg/dose Q6h for 7-10 days

and

- Gentamycin IV 5mg/kg/dose Q24h 5 days

→ If fever or pain, give

- Ibuprofen: 2-3mg/kg/dose Q8h or Paracetamol 10-15mg/kg Q6h, max 60mg/kg/day

Surgical

- Mastoidectomy
- Incision of abscess

→ When anaerobic infection is suspected: Add Metronidazole IV, 15-20 mg/kg/dose Q8h and culture sensitivity where possible

1.4.5. Epistaxis

Definition: Epistaxis is nose bleeding

Causes

- Local : Trauma, inflammation, foreign bodies, tumours of the nose and rhinopharynx, chronic using of nasal steroides, intra nasal growth like polyps
- Systemic : Cardiovascular diseases, blood diseases, liver diseases, kidney diseases, febrile diseases
- Upper respiratory disease : Sinusitis, allergic rhinitis
- Juvenile nasopharyngeal angiofibroma if profuse unilateral epistaxis associated with a nasal mass in adolescent boys
- Idiopathic (causes not known)

Signs and Symptoms

- Blood coming from the nose or the rhinopharynx
- History of recurrent nasal bleeding

Complications

- Hypovolemic shock
- Anaemia

Investigations (In complicated or recurrent cases)

- Full blood count, clotting time, bleeding time, prothrombin time
- CT scan and MRI if juvenile nasopharyngeal angiofibroma
- Other investigations should be requested based on general examination findings

Management

Non pharmaceutical

• Sit the patient up to avoid aspiration
• Cleaning of blood clots from the nose
• direct pressure applied by pinching the soft fleshy part of the nose applied for at least five minutes and up to 20 minutes
• Application of cold compresses on the nose
• Room humidifier
• Pack with ribbon gauze impregnated with topical ointments (Vaseline) and remove it after 12-24 hours

Pharmaceutical

•Application of a topical antibiotics ointment to the nasal mucosa has been shown to be an effective treatment for recurrent epistaxis

•Topical vasoconstrictor: Xylometazoline spray (otrivine) 0.5mg/ml
• Cauterization of the bleeding site with silver nitrate or 20% of solution trichloracetic acid under topical anesthesia
• Electro coagulation
• If severe bleeding with shock/or anemia, immediate blood transfusion is recommended
Recommendations
- Investigate for underlying causes
- Refer cases of severe and recurrent epistaxis
- Refer to ENT specialist for otolaryngologic evaluation if bilateral
bleeding or hemorrhage that did not arise from Kiesselback plexus persists

1.4.6. Sinusitis

Definition: Sinusitis is the inflammation of one or more sinus cavities.
Causes
- Rhinitis (most common cause)
- Trauma with open sinuses
- Bacterial infections: (Bacteria : S.pneumoniae, H. Influenza, Moraxella catarrhalis, staphylococcus Aureus, anaerobies)
- Viral: Common predisposing factors include: abscess and tooth extraction,chemical irritants, nasal polyp, deviation of nasal septum, perfumes or paint fumes, and changes in the weather

Signs and Symptoms
- Purulent nasal discharge (unilateral or bilateral)
- Fever and cough
- Nasal obstruction and congestion
- Frontal headache and heaviness of the head exaggerated on bending the head
- Persistant symptoms of upper respiratory tract infection
- On clinical examination, pressure on frontal and maxillary sinuses causes pain
- decreased sense of smell
- Periorbital oedema
- Anterior rhinoscopy shows pus coming through the middle meatus

Complications
- Local: Osteomylitis, orbital cellulitis, orbital abscess
- descending infections: pharyngitis, tonsillitis, bronchitis, pneumonia
- Systemic: septicemia, meningitis, brain abscess, thrombophlebitis of cavernous sinus, subdural empyema

Investigations
- Paranasal X-ray (shows opacification with air-fluid level)
- CT scan

Management
Medical treatment (consists of nasal decongestants and antibiotics)
• Treatment of first choice

→ Amoxicillin, Po 15-20mg/kg/dose Q8h 7-10 days
→ Paracetamol 10-15mg/kg/dose Q6hr
• Alternative treatment
→ Amoxicillin-clavulanate (amoxi-clav, augmentin®) 15-20 mg/kg/dose PO, Q8h 7 -10 days
→ Add Xylometazoline (Otrivine) 0.5% drops or simple argyrol drops 1% , 0.05%
Or
→ cefadroxyl : 25mg/kg/dose Q12h for 7 days
→ cefuroxime (Zinat): tabs 15mg/kg/dose Q12h for 7 days
→ Azithromycine 10mg/kg/dose Q24h for 3 days
→ Erythromycine 15-20 mg/kg/dose Q8h for 10 days
→ Rovamycine 3MI units: 50000-100000 UI/kg/dose Q8h for 10 days
→ Naphazoline-ephedrin nasal drops 2% 3 drop x3/day/7 days
Recommendation
- do not use nasal decongestants taking a monoamine oxidase inhibitor in hypertensive patient

1.4.7. Laryngitis

Definition: Laryngitis is inflammation involving the vocal cords and structures inferior to the cords
Cause
- Viral respiratory tract infection (Parainfluenza Virus Type 1 and 2, Rhinoviruses, Syncytial Viruses, adenoviruses)
Signs and Symptoms

- Progressive Laryngeal dyspnea
- Sore throat
- Hoarseness of voice
- Stridor
- Barking cough
- Fever
- Erythema and Edema of larynx

Complications

- Severe respiratory distress
- Secondary infection
- Airway obstruction

Investigation

- Unless there are signs of secondary infection

Management

Non Pharmacological management

- Humidified O2 therapy
- Plenty of fluids

Pharmacological treatment

- Adrenaline nebulisation 0.5ml/kg [of diluted 1:1000 (1 mg/ml)] in 3 ml Normal saline. Maximum dose 2.5ml for ≤ 4yrs old and maximum 5ml for > 4yrs old.
- Dexamethasone IM 0.3-0.6mg/kg per dose x 2/day/2days or Prednisolone PO 1-2mg/kg/day divided in 2 doses (Maximum dose 50mg in 24hrs)

Recommendation

- Patients who don't improve after treatment should be intubated

1.4.8. Epiglottitis

Definition: Acute epiglottitis is a life-treatening emergency due to respiratory obstruction. It is due to intense swelling of epiglottis and surrounding tissues with septic signs.

Cause

- It is caused by Haemophilus influenza type b. Since systematic vaccination, this condition has become very rare

	Croup	Epiglottitis
Onset	Over days	Over hours
Preceding coryza	Yes	No
Cough	Severe, barking	Absent or slight
Able to drink	Yes	No
Drooling saliva	No	Yes
Appearance	Unwell	Toxic, very ill
Fever	<38.5°C	>38.5°C
Stridor	Harsh, rasping	Soft, whispering
Voice, cry	Hoarse	Muffled, reluctant to speak

Management

- Urgent hospital admission and treatment
- Move the child only when ready for intubation under anesthaesia

- Intubation by senior anesthaesist, paediatrician and ENT in surgical room
- Urgent tracheostomy if intubation impossible
- Antibiotic treatment: cefotaxime IV 30-50 mg/kg/dose Q8h for 7-10 days

Or

- ceftriaxone IV 100mg/kg/dose Q24h for 7-10 days

1.4.9. Pertussis (Whooping Cough)

- Definition: this is a highly infectious form of bronchitis caused by
- bordetella pertussis. It has become rare since vaccination but it is endemic with epidemics every 3-4 years. Particular attention should be paid to young infants (before complete vaccination), adults (weaning effect of vaccine) and unvaccinated.
- Cause
- - Bordetella pertussis
- Signs and Symptoms
- - After one week of coryza (catarrhal phase), the child develops a characteristic paroxysmal cough followed by characteristic inspiratory whoop (paroxysmal phase, 3-6 weeks). It worsens at night with occasional vomiting.during paroxysm, the face goes red or blue and mucus flows from nose and mouth. It may cause apnea in young infants.

- The symptoms gradually decrease and may persists for months

(convalescent phase)

- Investigations
- - Culture if available
- - FBC: marked lymphocytosis (>15 *10^9/l)
- Management
- - Admit to hospital if infant (risk of apnoea)
- - Symptomatic treatment: O2, Naso-Gastro tube feeding
- - Erythromycin 15-20 mg/kg/dose Q8h for 14 days.
- Or
- - Azitrhomycin, Infants aged <6 months: 10 mg/kg/dose Q24h for 5 days.
- • Infants and children aged >6 months: 15 mg/kg (maximum: 500 mg) on day 1, followed by 10 mg/kg/dose Q24h (maximum: 250 mg) on days 2-5
- Recommendation
- - Prophylaxis for close contacts

- 1.4.10. Allergic Rhinitis

(a) (b)

(a) There is a habitually open mouth due to mouth breathing. **(b)** An allergic salute, from rubbing an itchy nose. (Courtesy Dr George Du Toit.)

- Definition: It is an inflammation of the mucous lining of the nose dueto hypersensitivity to inhaled allergens
- Causes
- - Allergy with common predisposing factors that include pollutin environment, dust, fumes, animals
- - Overuse of nasal decongestants (Rhinitis medicamentosa)
- - Viral infection
- - Bacterial infection secondary to viral infection
- Signs and Symptoms

- - Nasopharyngeal discomfort with nasal congestion
- - dry cough
- - Headache
- - Watery eyes
- - Sneezing and watery running nose
- - Sensation of nasal obstruction
- - Asthenia
- - Thick, sticky mucus (after 3-days)
- Complications
- - Otitis media
- - Sinusitis
- - Pharyngitis
- - Laryngo-bronchitis
- Investigations
- - Blood tests for allergens (Serum immunoassays for specific IgE)
- - Skin testing for specific allergens
- - Nasal smears for specific allergens
- Management
- - Avoid allergens
- - There is no cure for the common cold; treatment is given for symptom relief
- - Supportive care includes bed rest and drinking plenty of fluids
- Treatment of first choice
- • 2-5 years : chlorpheniramine tabs/syrup :1mg x3/day/1-3 days

- • 6-11years: chlorpheniramine tabs/syrup: 2mg x3/day/1-3 days
- • 12 years: chlorpheniramine tabs/syrup: 4mg x3/day/1-3 days
- • nasal steroids, 1-2 spray/nostril/dose Q12-24h
- • Avoid local nasal decongestants as they have long term side effects
- Alternative treatment
- • claritine tabs/syrup
- → Children 2 to 12 years of age: Body Weight > 30 kg, 10 ml [10 mg], (two 5 ml spoonful)
- → Children : Body Weight 30 kg; 5 ml [5 mg], (one 5 ml spoonful
- → Children 12 years of age and over: One tablet [10 mg] once daily or 2- 5 ml spoonful [10 mg] once daily
- Or
- • cetrizine
- → Children 6 months to <2 years: 2.5 mg (½ teaspoon) once daily
- → Children 2 to 5 Years: 2.5 mg (½ teaspoon) syrup once daily increased to a maximum dose of 5 mg per
- day given as 1 teaspoon syrup once a day or one ½ teaspoon syrup
- → Children 6 to 11 Years: 5 mg or 10 mg once daily depending on symptom severity

- → Children 12 Years and Older: 5 mg or 10 mg per day
-

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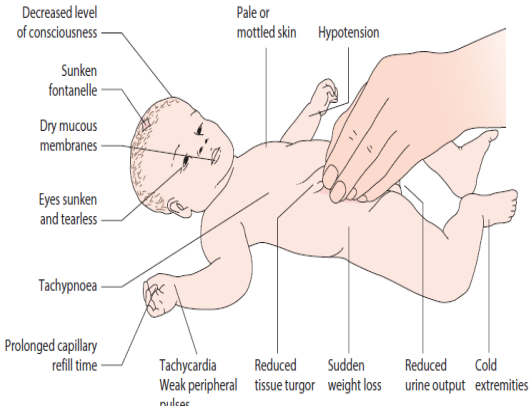
- **2. Gastro-intestinal Disorders**
  - 2.1. Acute Gastroenteritis

- Definition: Gastroenteritis is an inflammation of the stomach and
- intestines that causes diarrhea, vomiting, nausea and other symptoms
- of digestive upset. diarrhea is the passage of three or more loose or watery stools per day.
- It can be watery, bloody or containing mucus.
- Causes
- - Viral gastroenteritis: Rotaviruses are the most likely cause of
- infectious diarrhea in children under age 5
- - Bacterial gastroenteritis: Campylobacter, Salmonella or E. coli
- - Intestinal parasites: Giardia lamblia
- - Other causes include life threatening conditions that may be
- initiated by diarrhea: intussusceptions, appendicitis
- Signs and Symptoms
- CLINICAL EVALUATION OF DEHYDRATION
-

CLINICAL EVALUATION OF DEHYDR

Mild dehydration : 3 - 5% loss of body weight ***(Plan A)***	- No signs of dehyc
Moderate dehydration : 6-9% loss of body weight ***(Plan B)***	- Able to drink plu following: • Sunken E • Skin pinc • Restlessn
Severe dehydration : 10-15% loss of body weight ***(Plan C)***	- Pulse fine but un • Sunken E • Skin pinc

-
- Complications



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Clinical features of shock from dehydration in an infant.

-

- - Hypovolemic shock: (Tachycardia, cold hands, weak or absent pulse, capillary refill > 2 seconds, not alert)
- - Electrolytes imbalance: severe hyponatremia (<130mmol/L), severe hypernatremia (>150mmol/L), severe hypokalemia (<3mmol/L)
- - Cerebral œdema: (headache, convulsions, vomiting, nausea, weakness) due to rapid rehydration with hypotonic solutions
- - Intracerebral haemorrhage: due to severe dehydration in infants and young children
- Investigations
- - Stool exam: direct/culture (if blood or pus in stool)
- - FBC, CRP, Hemoculture if suspicion of bacterial blood stream
- - Electrolytes (Sodium and Potassium)
- - Glyceamia, urea/Creatinine if shock
- Note: Qualitative evaluation of dehydration (according to natremia)
- - Isotonic dehydration: Na 130 to 150 mmol/L
- - Hypertonic dehydration: Na > 150 mmol/L
- - Hypotonic dehydration: Na < 130 mmol/L
- Management
- - Admit the child: Absolute criteria of admission:

- • Profuse diarrhoea (> 8 stools/24h) with vomiting
- • Incoercible vomiting
- • Severe dehydration
- • Failure of home oral rehydration
- - If dehydration and shock without signs of malnutrition, give appropriate treatment as follows:
- • Consider CAB
- • 20ml/kg of normal saline (NS) or Ringers Lactate(RL) as quickly as possible IV Or PO in 15 minutes (see table below for estimation of required volume for 20ml/kg)
- • Repeat the bolus of NS or RL 3-4 times if persistence of signs of shock
- • Treat as severe dehydration after correction of shock
- Chapiter 2: Gastro-intestinal Disorders

- If severe dehydration without shock (Plan C):

Ringers Lactate (Normal Saline if unavailable)	Age < 12 months	Age ≥ 12 months to 5 years
Step 1	30 mls / kg over 1 hour	30 mls / kg over 30 mins
Step 2	70 mls / kg over 5 hours	70 mls / kg over 2.5 hours
Then re-assess child, if signs of severe dehydration persists repeat step 2. If signs improve treat for moderate dehydration		

-

- If moderate dehydration (Plan B):

• Give ORS 75ml/kg during 4 hours

After 4 hours:

• Reassess the child and classify the child for dehydration

• Select the appropriate plan to continue treatment

• Begin feeding the child in clinic

HOW TO ADMINISTER ORS

HOW TO ADMINISTER ORS

By bottle	- Give 1/3 during 1st hour, then 2/3 during 3 following hours. *E.g.: 10 kg - dehydrated 7%. Should receive 75 ml/kg = 750 ml SRO in 4 hours* - Give 60 ml every 15 min during 1st hour - Then 170 ml every hour during 3 following hours
Spoon or Seringues	- Give 5 ml every 1 to 2 min → 300 to 150 ml in 1 hour (Very efficacious if vomiting +++ and give important volumes)
Naso-gastric tube	- If vomiting +++ - If fatigue +++

If the mother must leave before completing treatment:

- Show her how to prepare ORS solution at home

- Show her how much ORS to give to finish 4-hour treatment at home
- Give her enough ORS packets to complete rehydration
- Explain the 4 rules of home treatment:

→ Give extra fluid: Give the child more to drink as is wanted by the child

→ Give Zinc supplements for 10–14 days:

 < 6 months: 1/2 tablet (10 mg) per day, ≥ 6 months:1 tablet (20 mg) per day

→ Continue feeding: Initial 4-hour rehydration period, breastfed children should continue to breastfeed frequently throughout

→ Return the child to the health facility if :

- drinking poorly or unable to drink or breastfeed
- Becomes more sick
- develops fever
- Has blood in the stool

- If no dehydration (Plan A):

• Treat the child as an outpatient; give ORS 10ml/kg after each watery stool

- Antidiarrhoeal medications
  - The locally available diosmectite (Smecta®) has been shov and effective in reducing stool output and duration of diar by restoring integrity of damaged intestinal epithelium, al bind to selected bacterial pathogens and rotavirus.
    Other anti diarrhoeal agents like kaolin (silicates), loperan motility) and diphenoxylate (anti motility) are not recomn
- Antiemetic medication
  - Not recommended, potentially harmful.
- Probiotics
  - Probiotics has been shown to reduce duration of diarrhoe randomized controlled trials. However, the effectiveness i and dose specific. Therefore, only probiotic strain or strair proven efficacy in appropriate doses can be used as an ad standard therapy.

• Counsel the mother on the 4 rules of home treatment:

→ Give extra fluid
→ Give zinc supplements
→ Continue feeding
→ Give advice on when to return for review

Particular forms of dehydration

Type	Intervention	Comment
Hyponatremia (Na < 130mmol/L)	**Na Deficit = 0.6 x W in kg x** (Na^+_d - Na^+_m) during 4 hours **W**= weight **d** = desired sodium **m** = measured sodium	Do not correct too quickly to avoid CNS lesion
Hypernatremia (Na > 150mmol/L)	Slowly correct dehydration over 48 hours	Risk of convulsions in case of rapid correction
Hypokalemia	If Potassium< 2.5 mmol/L give KCl 30-40 mmol/L/24hours	Give KCl

2.2. Persistent Diarrhea

Coeliac

disease causing wasting
of the buttocks and
distended abdomen.

Definition: Persistent diarrhea is a diarrhea, with or without blood,
which begins acutely and lasts for 14 days or longer.

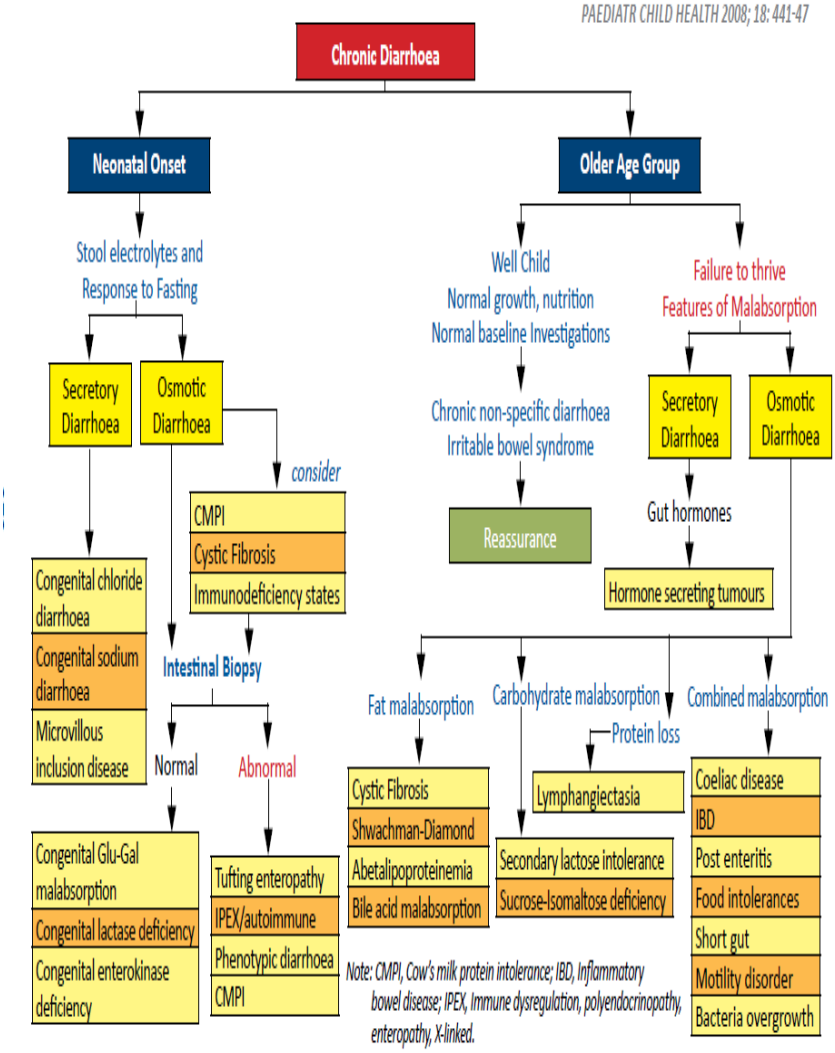
AGE	ETIOLOGIES
Infancy	- Post gastroenteritis mal-absorption syndrome - Cow's milk/soy protein tolerance - Secondary disaccharidase deficiencies - Cystic fibrosis
Childhood	- Secondary disaccharidase defiencies - Giardiasis - Post-gastroenteritis mal-absorption syndrome - Celiac disease - Cystic fibrosis - HIV - Malnutrition
Adolescence	- Irritable Bowel Syndrome - HIV - Inflammatory Bowel Disease

Differentiation of Osmotic from Secretory Diarrhoea		
Parameter	**Osmotic diarrhoea**	**Secretory diarrhoea**
Stool volume	Small (generally <200ml/24 hours)	Large (>200ml/24 hours)
Response to fasting	Diarrhoea stops	Diarrhoea continues
Stool Osmolality	> (Stool Na + K) x 2	= (Stool Na + K) x 2
Osmotic Gap	> 135 mOsm/l	< 50 mOsm/l
Stool Sodium	< 70 mmol/l	> 70 mmol/l
Stool Potassium	< 30 mmol/l	> 40 mmol/l
Stool Chloride	< 35 mmol/l	> 40 mmol/l
Stool pH	<5.5	> 6.0
Stool reducing substance	Positive (>0.5%)	Negative
Adapted from M Ravikumara. Investigation of chronic diarrhea. Paediatrics and Child Health 2008; 18: 441-47		

Investigations in Chronic Diarrhoea
Baseline investigations
Stool microscopy ova, cysts, parasites, fat globules
Stool microbiology
Stool pH, reducing substances, electrolytes
Full blood count and differential
Urea and electrolytes, CRP and ESR
Liver function tests including albumin
Coeliac serology
Subsequent investigations
Stool elastase-I
Stool alfa-1-antitrypsin
Vitamins A, D, E, coagulation, B12, folate levels, Ca, Mg, phosph
Endoscopy, colonoscopy and biopsies for histology, disacchari bacterial culture, Electron microscopy
Imaging studies x-ray, ultrasound, barium, MRI
Sweat test
Immunoglobulins, subclass, lymphocyte and neutrophil functic complements
Zinc level
Cholesterol, triglycerides, low-density lipoproteins
Autoantibodies including anti-enterocyte antibodies
Isoelectric focussing of transferrin
Gastrin, secretin, calcitonin, VIP
Manometric studies
Urinary laxatives
Breath hydrogen tests
Plasma and urinary bile acids and salts
Response to dietary modifications
Adapted from M Ravikumara. Investigation of chronic diarrh and Child Health 2008; 18: 441-47

ALGORITHM FOR APPROACH TO CHRONIC DIARRHOEA

ADAPTED FROM RAVIKUMARA, PAEDIATR CHILD HEALTH 2008; 18: 441-47



Note: CMPI, Cow's milk protein intolerance; IBD, Inflammatory bowel disease; IPEX, Immune dysregulation, polyendocrinopathy, enteropathy, X-linked.

Complications
- dehydration
- Failure to thrive, malnutrition
- Immunosuppressant

Investigations (Will vary according to the suspected etiology)
- Stool examination: PH, White blood count, Fat, Ova, osmolarity, Culture
- FBC, CRP, electrolytes, urea and creatinine
- Sweat chloride if suspicion of cystic fibrosis
- Barium study
- Small bowel biopsy
- Endoscopy: Sigmoidoscopy or coloscopy with biopsy

Management
- Oral rehydration
- Treat the cause (see algorithm)

2.3. Bloody Diarrhea

Definition: Frequent (>3/day) passage of blood and/or mucus in the stool

Causes
- Amoebic dysentery is the most common serious cause in children
- Bacterial infections (e.g. Shigella, salmonella)
- Parasitic infestations (e.g. amoebic dysentry)
- Milk allergy
- Chronic inflammatory bowel disease

Signs and Symptoms
- Sudden onset

- Abdominal cramps
- Peritonism urgency, fever and diarrhea with blood and mucus in the stool
- meningismus and convulsions may occur
- Exclude intussusceptions which includes:
• pain or abdominal tenderness
• bile-stained vomitus
• red currant jelly-like mucus

Complications
- dehydration
- Convulsions
- Shock
- Toxic megacolon
- Acidosis
- Rectal prolapse
- Renal failure
- Haemolytic uraemic syndrome

Investigations
- Stool culture to confirm diagnosis of Shigellosis
- Stool microscopy reveals many polymorphs and blood
- Immediate microscopy of warm stool to diagnose amoebic dysentery

Non-pharmacological
• Ensure adequate nutrition and hydration

Pharmacological
• Fluid and electrolyte replacement (see Acute diarrhea)
• ciprofloxacin, oral, 15 mg/kg/dose 12 hourly for 3 days
Or

• ceftriaxone, IV, 20–80 mg/kg as a single daily dose for 5 days (If hospitalised or if unable to take oral antimicrobial agents)
• Metronidazole, oral, 15 mg/kg/dose 8 hourly for 7 – 10 days (If amoebic dysentery, seen on stool microscopy)

Recommendation

- Refer patient to the specialist, if dysentery with complications, e.g. persistent shock, haemolytic uraemic syndrome and toxic megacolon

2.4. Constipation

Definition: Constipation is an acute or chronic condition in which bowel movement occurs less often than usual or consist of hard, dry stool that are painful or difficult to pass.

Causes

- Lack of exercise
- Certain medicines
- Metabolic, endocrine, neurogenic and lower bowel abnormalities
- Psychogenic disorders
- Chronic use of enemas
- Not drinking enough water
- diet that does not include an adequate amount of fiber-rich foods
- Anal fissure (a tear or crack in the lining of the anus)
- Chronic kidney failure
- Hischprung disease
- Colon or rectal cancer

- depression
- Hypercalcemia (abnormally high levels of calcium in the blood)
- Hypothyroidism (underactive thyroid gland)
- Illness requiring complete bed rest
- Irritable Bowel Syndrome
- Stress

Signs and Symptoms

- Symptomatic bowel impaction
- Blood in the stool
- Changes in bowel patterns
- Abdominal pain, distension

Complications

- Bowel obstruction
- Chronic constipation
- Hemorrhoids
- Hernia
- Spastic colitis
- Laxative dependency

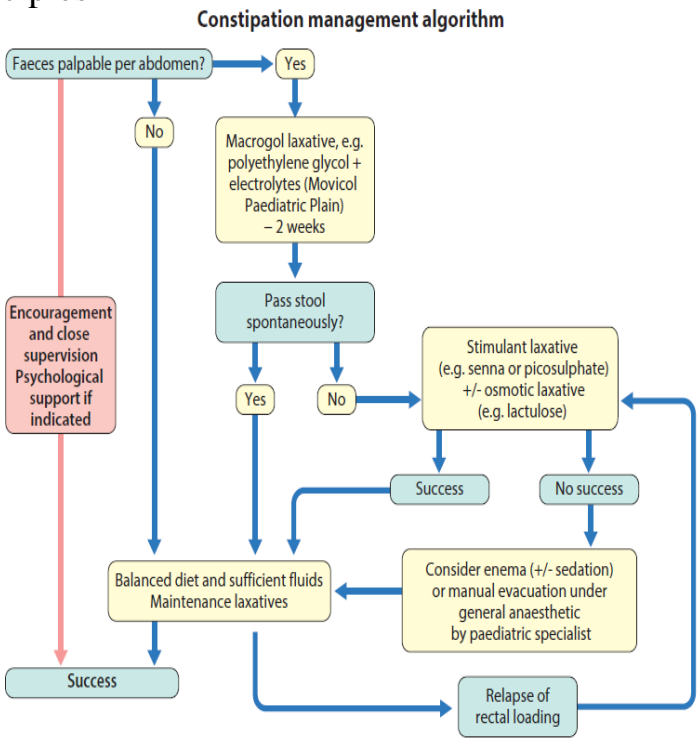
Investigations

- Abdominal X-ray
- Barium meal - reveals blockage inside the intestine in particular cases
- Laboratory analysis of blood and stool samples for internal bleeding

- Sigmoidoscopy (examination of the sigmoid area of the colon
with a flexible tube equipped with a magnifying lens), rarely
indicated.

Management

Principles



- Treatment involves 3 steps:

→ Initial clearance of stool

→ Prevent reaccumulation of hardened retained stool (diet change with additional natural fibre from fruit, vegetables and bran)
→ Retraining of the gut to achieve regular toilet habits
Management is long-term, and requires the active involvement of the parents
Pharmacological
• Enema twice daily for 3 days for faecal clearance if faecal loading
• Lactulose (Duphalac) for 1 week but if 3 stools are passed/day stop it
• Bowel re-training
• In refractory cases:
→ Lactulose, oral, twice daily
■ < 1 year 2.5 mL
■ 1–6 years 5 mL
■ > 6 years 10 mL
• determine and treat the underlying cause
Recommendations
- Refer patient to the specialist, if an organic cause e.g. constipation from birth in a breast-fed baby is suspected
- If faecal loading continues, maintenance therapy should be continued for months to years

2.5. Constipation and Encopresis

Definition: Constipation is the delay or difficulty in passage of stools during defaecation that has been present for 2 weeks or longer. Stoolis usually hard.

Encopresis also known as faecal soiling is the involuntary leakageof small amounts of soft or watery stool in a child with chronic constipation.

Causes

- Psycho social precipitants
- Functional (incorrect diet, lack of exercise, poor fluid intake)
- Metabolic or Neurological Abnormalities
- Endocrine abnormalities (Hypothyroidism)
- Chronic use of laxatives
- Obstructive lesions (acquired and congenital defects)

Signs and Symptoms

- Abdominal pain often associated with encopresis
- Infrequent defecation
- Pain or strain on defecation
- Hard stool
- Feeling of incomplete evacuation (Tenesmus)

Complications

- Anal fissure, ulcers and prolapse
- Over flow incontinence (Encopresis)
- Stasis syndrome with bacterial overgrowth

Investigations

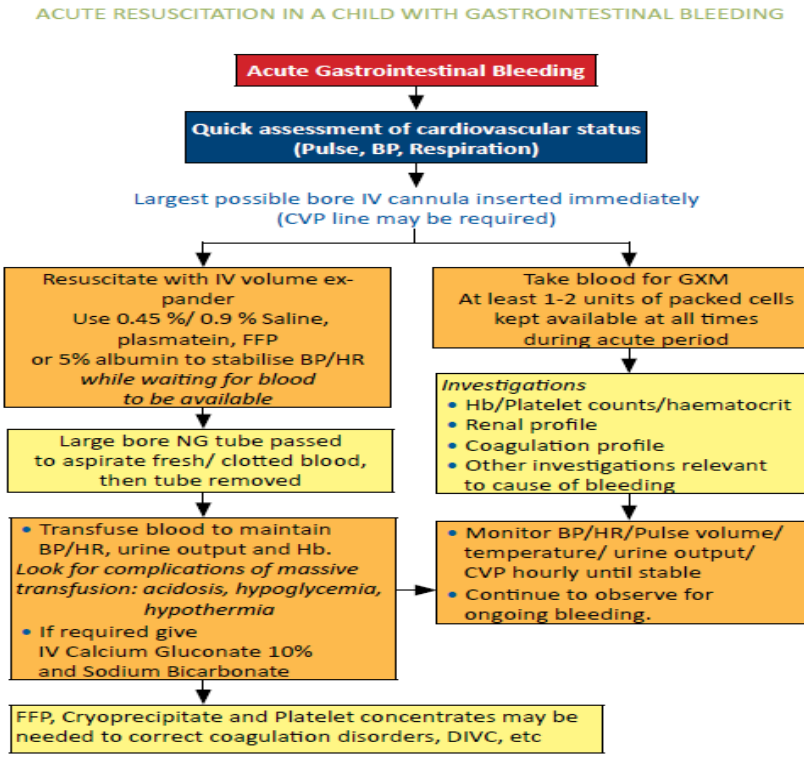
- Barium Enema
- Abdominal x-ray in suspected obstructive lesions
- Thyroid function tests when indicated
- Stool analysis
- Investigate other functional lesions

Management
Non-pharmacological management
• Rehydrate to increase fecal bulk and soften stool
• Education of patients/parents on diet, exercise, etc.
• diet change with additional natural fibre from fruit and vegetables
• Treatment involves 3 steps:
→ Initial clearance of stools
→ Prevent re-accumulation of hardened retained stool
→ Retraining of the gut to achieve regular toilet habits
Pharmacological management
• Glycerin Suppositories 1 suppo/dose according to occurrence of symptoms
Or
• Lactulose syrup <1 yr: 5-10ml/24 hr PO Od; 1-6 Yrs 10-20 ml/24 hours PO Od; 7-14 yrs 20-50ml/24 hrs PO Od
Or
• Bisacodyl (Dulcolax) 0.3mg/kg/day PO Od maximum dose 30mg/24 hours
Recommendations
- Refer to tertiary health facility in cases of inadequate response to therapy for further investigation
- If continued constipation therapy should be continued for months to years

2.6. Upper Gastro-Intestinal Tract Bleeding

Definition: Upper gastrointestinal bleeding (arising proximal to the ligament of Treits in the distal

duodenum) commonly manifested by hematemesis and/or melena.



Decision making after acute resuscitation
Reassessment of patients
When patient's condition is stable and resuscitative measures have been instituted, *Assess patient for cause of bleeding and the need for surgery.*
History is reviewed. Ask for history of chronic liver disease, dyspepsia, chronic or intermitte gastrointestinal bleeding (e.g. polyps), drug ingestion (anticoagulants, aspirin), or acute fever (dengue haemorrhagic fever), easy bleeding tendencies, antibiotics treatment (pseudomembranous colitis).
Physical examination should be directed towards looking for signs of chronic liver disease (spider angiomata, palmar erythema, portal hypertension or splenomegaly) or telengiectasia / angiomata in mouth, trunk, etc.)
Diagnostic measures to localise source of bleeding
• *Oesophagogastro-duodenoscopy (OGDS)* or colonoscopy can be performed when patient's condition is stable.
• Double contrast barium study less useful than endoscopy but may be indicated in patients when endoscopy cannot precisely locate the source of bleeding (e.g. in intussusception).
• Visceral angiography can precisely locate the source of bleeding. But is only reserved for patients with a difficult bleeding problem.

Definitive measures to management of gastrointestinal bleeding
Medical Cause
Bleeding peptic ulcer • Start H2 receptor antagonist (e.g. cimetidine or ranitidine). Proton pump inhibitor (omeprazole) should be considered when available as it has higher acid suppressant activity. Pantoprazole infusion has been increasingly used "off label" (discuss with Paediatric Gastroenterologist). • If biopsy shows presence of *Helicobacter pylori* infection, treat accordingly. • Stop all incriminating drugs e.g. aspirin, steroids and anticoagulant drugs if possible.
Bleeding oesophageal varices or ulcer • Do not transfuse blood too rapidly as this will lead to increase in CVP and a rapid increase in portal pressure will precipitate further bleeding. • Aim to maintain Hb at 10 g/dL. • Refer Paediatric Surgeon and Paediatric Gastroenterologist to consider use of octreotide.
Pseudomembranous colitis • Stop all antibiotics • Start oral metronidazole or oral vancomycin immediately.
Surgical Cause
When surgical cause is suspected, early referral to the surgeon is important so that a team approach to the problem can be adopted. • Intussusception requires immediate surgical referral and intervention. • Meckel's diverticulum • Malrotation

Management

Main objectives

- Relieve or treat hemorrhagic shock if present
- Stop bleeding
- Treat the causative agent

Emergency treatment

- CAB (include Blood transfusion if necessary
- Assess to causative agent and treat according if need of endoscopy then refer to centre where it's available

Pharmacological Management according to age

- Neonates

→ cimetidine IV 5-20mg/kg divided in 2 doses

OR

Ranitidine IV 2mg/kg/24 divided in 2-3 doses for 10 days

Or

→ Omeprazole, PO 0.5–1 mg/kg, 12– 24 hourly for 10 days

- Infants and toddlers

→ Octreotide, IV bolus, 1–2 mcg then 1–5 mcg/kg/hour by infusion, initiated by the specialist in case of cases of variceal bleeding (difficult to control, to help control bleeding before endoscopy, or when endoscopy is unsuccessful, contraindicated, or unavailable)

→ Omeprazole, PO

- 1 month–2 years: 2.5mg, 12 hourly
- 2–6 years 5 mg, 12 hourly initiated by the specialist for post bleed prophylactic management

- Older children and adolescents

→ Omeprazole, PO

- < 20 kg: 10 mg Qd
- >20 kg: 20 mg Qd

Note: *Endoscopy is recommended to be performed within 24 to 48hours for infants and children presenting with UGI bleeding*

that is acute and severe, it can be performed for diagnosis andtreatment (sclerotherapy in oesophageal variceal)

Alternative treatment

- Propranolol oral, 2–8 mg/kg/24 hours in 3 divided doses (to reduce the pulse rate by 25%)
- Surgical over sewing if endoscopy and sclerotherapy or banding have failed

Recommendations

- Refer all cases to the specialist for appropriate diagnosis and treatment
- Refer all bleeding varices - after commencement of resuscitation and octreotide, if available

2.7. Peptic Ulcer Disease

Definition: This refers to ulceration of gastric or duodenal mucosa that tends to be chronic and/or recurrent.

Cause

- Helicobacter pylori (H. pylori) - in developing nations, the

majority of children are infected with H. pylori before the age of 10 and adult prevalence peaks at more than 80 percent before age 50

Signs and Symptoms

- Peptic ulcers may be present with dyspeptic or other gastrointestinal symptoms or may be completely asymptomatic,

sometimes until complications such as hemorrhage or perforation occur. The symptoms associated with peptic ulcers are not sensitive or specific and the differential diagnosis is broad.

- Most common: Ulcer-like or acid dyspepsia (burning pain; epigastric hunger-like pain; relief with food, antacids, and/or anti secretory agents). - Food-provoked dyspepsia or indigestion (postprandial epigastric discomfort and fullness, belching, early satiety, nausea, and occasional vomiting): food-stimulated acid secretion persists for three to five hours; thus, classic dU symptoms occur two to five hours after meals.
- Reflux-like dyspepsia

Complications

- The natural history of peptic ulcer ranges from resolution without intervention to development of complications: acute or Chronic blood loss or perforation
- Iron deficiency anaemia

Investigations

- Stool analysis for occult blood
- FBC
- For HP:

• It is recommended that the initial diagnosis of H. pylori infection be based on positive histopathology plus positive
rapid urease test, or positive culture.

• A validated ELISA for detection of H. pylori antigen in stool is a reliable non-invasive test to determine whether H.
pylorus has been eradicated.
• Tests based on the detection of antibodies (IgG, IgA) against H. pylori in serum, whole blood, urine and saliva are not reliable for use in the clinical setting.

Note: *Specialists recommend: In children with refractory iron deficiency anemia, where other causes have been ruled out, testing for H. pylori infection may be considered (grade of evidence - low)*

Management
- Avoid any foods that cause pain to the patient (e.g. acid foods, soda drinks)
- Avoid gastric irritating drugs (NSAIds)
- Give magnesium-based antacids or combined magnesium-
AluminiumFirst line H pylori eradication regimens are:
• Triple therapy with:
→ PPI + Amoxicillin + Imidazole
Or
→ PPI + Amoxicillin + clarithromycin
Or
→ Bismuth salts + Amoxicillin + Imidazole
Or
• Sequential Therapy Triple therapy for eradication of H. pylori (duration: 10 – 14 days)

→ Omeprazole PO
- 15-30 kg: 10 mg twice daily
- >30 kg: 20 mg twice daily

Or

→ cimetidine 20–40mg/kg/day + clarithromycin : 500mg BId + Amoxicillin 1g twice daily

Or

→ cimetidine 20–40mg/kg/day +Clarithromycin : 500mg + Metronidazole 500 mg (15–20mg/kg/day) twice daily

Note: *A reliable non-invasive test for eradication is recommended at least 4 to 8 weeks following completion of therapy*

Recommendations
- Refer to a specialist, if there is severe hemorrhage
- Stabilize the patient before transfer
- Infuse IV fluids/blood to maintain normal volume/pulse
- Ensure continuous assessment of further blood loss (Persistent tachycardia, postural hypotension, continuing haematemesis)
- definitive treatment / eradication of H. pylori

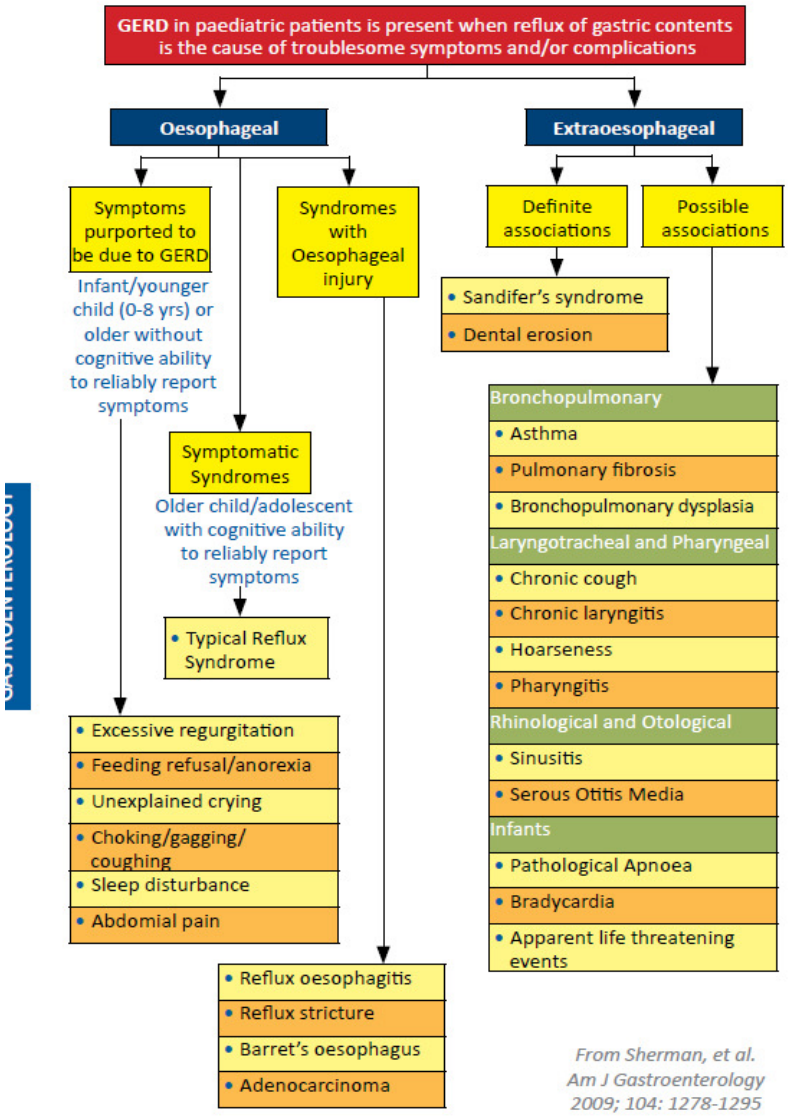
- 2.8. Gastroesophageal Reflux

A pH sensor has been placed in the lower oesophagus.

-Definition: GER is the passage of gastric contents into the esophagus with or without regurgitation and vomiting. GER is a normal physiologic process occurring several times per day in healthy infants, children, and adults.

- Most episodes of GER in healthy individuals last <3 minutes, occur in the postprandial period, and cause few or no symptoms. In contrast, Gastroesophagial reflux disease GERd is present when the reflux of gastric contents causes troublesome symptoms and/or complications.

GLOBAL DEFINITION OF GERD IN THE PAEDIATRIC POPULATION



From Sherman, et al.
Am J Gastroenterology
2009; 104: 1278-1295

- Causes and risk factors
- - The cause is still unclear
- - Anatomical abnormalities such as a hiatal hernia
- - Long term use of nasal gastric tube
- - diet that stimulates gastric acid production
- - Neurologic impairment (NI), obesity, certain genetic syndromes,
- esophageal atresia (EA), chronic lung diseases, and those with a history of premature birth
- Signs and Symptoms
- In infants and toddlers, there is no symptom or symptom complex that is diagnostic of GERd or predicts response to therapy. In older children and adolescents, as in adult patients, history and physical examination
- may be sufficient to diagnose GERd if the symptoms are typical. The following is suggestive:
- - In newborn: Recurrent vomiting, stridor, apnea
- - In infant: Recurrent vomiting, respiratory manifestations, (dry cough, recurrent wheeze or cough, chronic obstructive airway disease) recurrent aspiration pneumonia, stridor, apnea

In children /adolescent: Heartburn, epigastria or chest pain.

- Respiratory manifestations: (dry cough, recurrent wheeze or cough, chronic obstructive airway disease
- Complications
- - dysphagia
- - Odynophagia
- - Weight loss
- - Anemia
- - Esophagitis
- - Aspiration pneumonia
- - Barrett's esophagus
- - Abnormal posturing or opisthotonus (Sandifer Syndrome)
- Investigations (when GER is persisting despite basic management)
- - 24 hours esophageal PH monitoring
- - Endoscopy with biopsy to rule out oesophagitis
- - Barium X-rays for severity of oesophagus stenosis
- - FBC look for anemia
- Management
- Non-pharmacological
- • Postural treatment: Prone and lateral positions are associated with an increased incidence of sudden infant
- death syndrome (SIdS). The risk of SIdS outweighs the benefit of prone or lateral sleep

position on GER; therefore, in most infants from birth to 12 months of age, supine positioning during sleep is recommended.

- • dietary measures such as thickened food if not breastfeeding, frequent small volume of solid foods
- Pharmacological
- • Less Severe or Non Erosive
- → Anti-acids:
- ■ Sodium alginate (Gaviscon Enfant) /antacid
- combination, oral, month 1ml after each meal
- o 1-2 months 1.5 mls after each meal
- o 2-4 months 2mls after each meal
- → Aluminium and Magnesium hydroxide (Maalox)
- Syrup 0.5 ml/kg/dose PO QId
- → H2 Antagonists:
- ■ cimetidine IV/syrup/tab
- o Neonates: 5-20mg/kg/24 hr divided in 2 doses
- o Infants: 10-20 mg/kg/24hrs divided in 2 doses
- o Children: 20-40mg/kg/24hr divided in 2 doses
- •Severe or Erosive
- → Omeprazole, oral
- ■ Neonate 0.5–1 mg/kg, 12– 24 hourly
- ■ Children 1- 16 years

- o 5 kg to <10 kg: 5 mg once daily
- o 10 kg to ≤20 kg: 10 mg once daily
- o >20 kg: 20 mg once daily
- Alternate dosing: 1 mg/kg/dose once or twice daily; higher doses may be necessary in children between 1-6 years
- → Add
- ■ Pro-Kinetics: Domperidone (Motilium) 0.3 – 0.6 mg/kg/24hrs PO divided in 3 doses (TdS).
- Maximum 30mg/24 hours
- → ANd
- ■ Metoclopromide IV/IM/PO 0.1-0.2mg/kg/dose
- TdS. Maximum dose 0.5mg/kg/24 hours
- Recommendations
- - Refer to tertiary level gastro-oesophageal reflux not responding to treatment
- - Educate parents/guardians on patient diet
- - Eat small, frequent meals

2.9. Tropical Splenomegaly (Hyperreactive malarious splenomegaly) (HMS)

- Definition: It is a massive enlargement of the spleen resulting from
- abnormal immune response to repeated attacks of malaria
- Signs and Symptoms
- - Chronic abdominal swelling and pain.
- - Weight loss

- - Intermittent fever
- - Some patients present with anaemia, generalized weakness,
- cough, dyspnea, epistaxis, headache, increased skin and respiratory infections
- Clinical diagnosis
- - Splenomegaly of at least 10cms
- - Regression of the spleen by at least 40% by 6 months on antimalarial therapy.
- Complications

- Hypersplenism leading to anemia, leukopenia and thrombocytopenia, bleeding
- Splenic lymphoma
- - death
- Investigations
- - Blood smear
- - Complete blood count (for Hb, Platelets)
- - Serum levels of IgM (at least 2Sd above normal limit)
- Management
- Pharmacological treatment
- • Doxycycline tabs /day for 6 months
- → Children >8 years (<45 kg): 5 mg/kg/day Od
- → Children >8 years (>45 kg): treat as adults
- Or

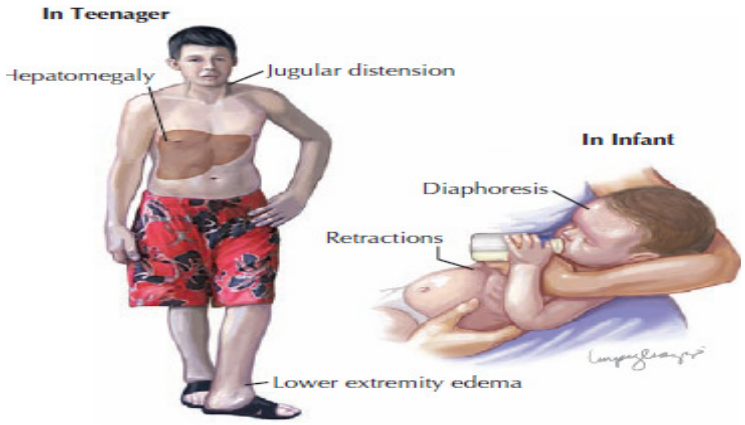
- • Mefloquine 5mg/kg weekly without exceeding 250mg/week of adult dose for 6 months

Dr: Essam Abdullah 01123232188

-

- **3. Cardiovascular Diseases**

- Most cardiac diseases in young children are congenital, while those in
- older children may be acquired or congenital.
  - **3.1. Heart Failure (Congestive Cardiac Failure)**



Clinical presentation of heart failure in a teenager and infant.

-

- Definition: It is a clinical syndrome reflecting the inability of the myocardium to meet the oxygen and nutritional metabolic requirements of the body.
- Causes

Box 17.2 Causes of heart failure

1. Neonates – obstructed (duct-dependent) systemic circulation

- Hypoplastic left heart syndrome
- Critical aortic valve stenosis
- Severe coarctation of the aorta
- Interruption of the aortic arch

2. Infants (high pulmonary blood flow)

- Ventricular septal defect
- Atrioventricular septal defect
- Large persistent ductus arteriosus

3. Older children and adolescents (right or left heart failure)

- Eisenmenger syndrome (right heart failure only)
- Rheumatic heart disease
- Cardiomyopathy.

-

Causes of Heart Failure	
Congenital heart disease	Acquired valvular disease
Left to right shunt lesions	• Chronic rheumatic valvular diseases
• VSD, PDA, AVSD, ASD	• Post infective endocarditis
Obstructive left heart lesions	Myocardial disease
• Hypoplastic left heart syndrome,	*Primary cardiomyopathy*
• Coarctation of aorta, aortic stenosis	• Idiopathic, familial
Common mixing unrestricted pulmonary flow	*Secondary cardiomyopathy*
• Truncus arteriosus, TAPVD, tricuspid atresia with	• Arrhythmia-induced: congenital heart block, atrial ectopic tachycardia
• TGA, single ventricle, pulmonary atresia with VSD,	• Infection: post viral myocarditis, Chagas disease
• Large aortopulmonary collateral	• Ischaemic: Kawasaki disease
Valvular regurgitation	• Myopathic: muscular dystrophy,
• AV valve regurgitation, Ebstein anomaly	• Pompe disease, mitochondrial dis.
• Semilunar valve regurgitation	• Metabolic: hypothyroidism
Myocardial ischaemia	• Drug-induced: anthracycline
• Anomalous origin of left coronary artery from pulmonary artery.	• Others: iron overload (thalassaemia)
	Acute myocarditis
	• Viral, rheumatic, Kawasaki disease

-

Clinical presentation

• Varies with age of presentation.

• ***Symptoms of heart failure in infancy:***

- ***• Feeding difficulty: poor suck, prolonged time to feed, sweating during feed.***

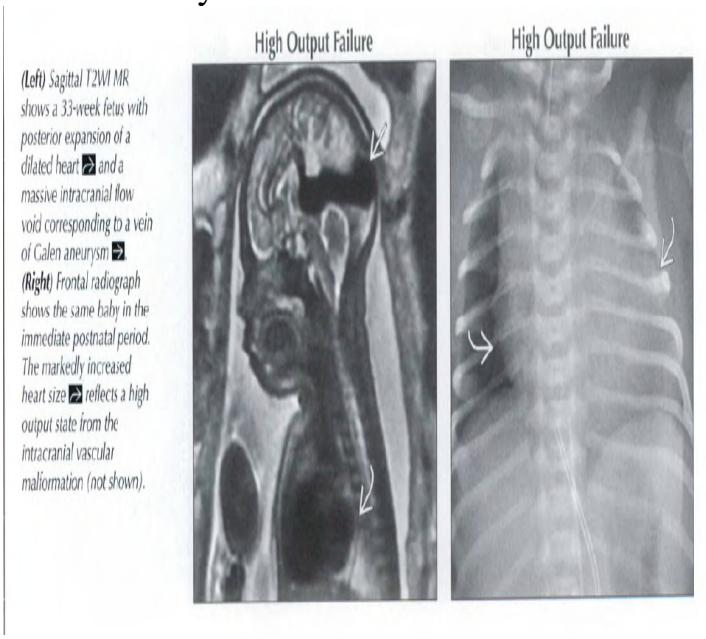
• ***Signs of heart failure in infancy:***

• ***Resting tachypnoea, subcostal recession.***

• ***Tachycardia, Poor peripheral pulses, poor peripheral perfusion.***

• Hyperactive praecordium, praecordial bulge.
• Hepatomegaly.
• Wheezing.
• Common signs of heart failure in adults, i.e. increased jugular venous
pressure, leg oedema and basal lung crackles are not usually found in children.
• Recurrent chest infections.

- ***• Failure to thrive.***
- Investigations
- - FBC, Electrolytes, Urea and Creatinine, Blood Gas if available
- - Chest X-ray
-



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- ECG
- - Echocardiogram

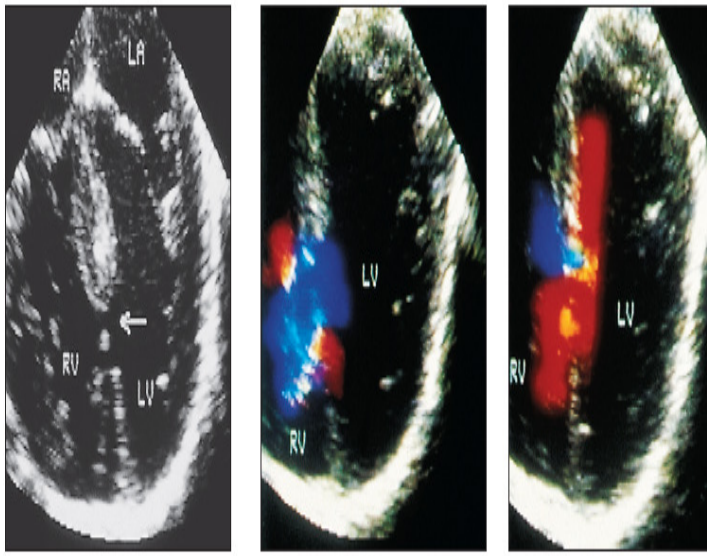


Figure 17.4 (a) Echocardiogram showing a medium-sized muscular ventricular septal defect (arrow). (b) The colour Doppler shows a left-to-right shunt (blue) during systole. (c) There is also a small right-to-shunt (red) during diastole (RA, right atrium; LA, left atrium; RV, right ventricle; LV, left ventricle).

- Management
- Non pharmacological
- • Oxygen therapy
- • Semi- Sitting position (cardiac bed)
- • Restrict fluids to 2/3 of maintenance (aim at urine output of 2ml/kg/h)
- • Low sodium diet
- • Strict bed rest
- • Ensure adequate nutrition

- • Recognize and treat the underlying conditions e.g. fluid overload, hypertension infection
- • Monitoring of vital signs: RR, HR, BP, O2 saturation, urine output
- Pharmacological

Antifailure medications

• Frusemide (loop diuretic) Dose: 1 mg/kg/dose OD to QID, oral or IV

• Continuous IV infusion at 0.1 – 0.5 mg/kg/hour if severe fluid overload

• Use with potassium supplements (1 - 2 mmol/kg/day) or add potassium sparing diuretics.

• Spironolactone (potassium sparing diuretic, modest diuretic effect) Dose: 1 mg/kg/dose BD

• Captopril Angiotensin converting enzyme inhibitor, afterload reduction agent

• Dose: 0.1 mg/kg/dose TDS, gradual increase up to 1 mg/kg/dose TDS

• Monitor potassium level (risk of hyperkalaemia)

• Digoxin

• Role controversial

• Useful in heart failure with excessive tachycardia, supraventricular tachyarrhythmias.

• IV inotropic agents - i.e. Dopamine, Dobutamine, Adrenaline, Milrinone

• Use in acute heart failure, cardiogenic shock, post-op low output syndrome.

Specific management

• Establishment of definitive aetiology is of crucial importance

• Specific treatment targeted to underlying aetiology.

Examples:

• Surgical/transcatheter treatment of congenital heart lesion.

• Pacemaker implantation for heart block.

• Control of blood pressure in post-infectious glomerulonephritis.

- • High dose aspirin ± steroid in acute rheumatic carditis.

- **3.2. Cardiogenic Shock**

- Definition: It is a dramatic syndrome characterized by inadequate
- circulatory provision of oxygen due to cardiac pump failure secondary to poor myocardial function, so that the metabolic demands of vital organs and tissues are not met.
- The patient often has a known case of heart disease with signs of heart failure but may also be a new case with heart failure.
- Signs and Symptoms
- - Hypotension
- - Tachycardia

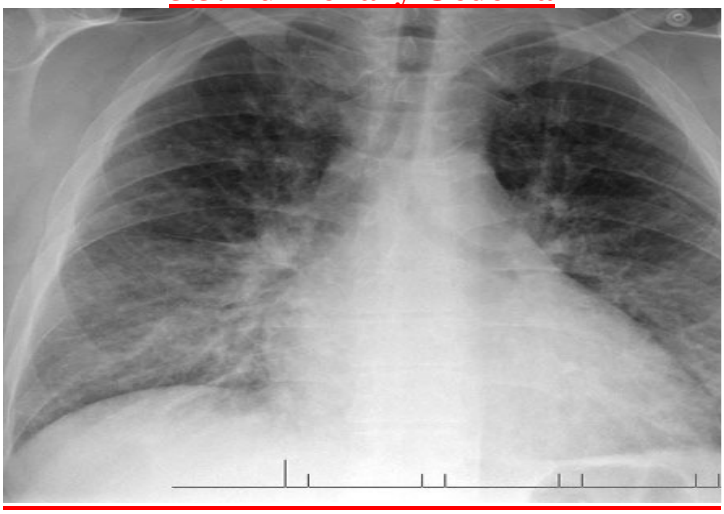
- - Gallop rhythm
- - Hepatomegaly
- - Crackles/wheezes
- - Weak and fast pulses (or absent)
- - Cold extremities/ palor
- - Capillary refill > 2 seconds
- - Oliguria/anuria
- Management
- Non pharmacological management
- • Avoid excessive IV fluids, the patient is fluid overloaded in this case, give 2/3 of maintenance (aim at urine output of 2ml/kg/h)
- •Oxygen therapy: 10-15l/min with mask and reservoir bag
- • Semi- Sitting position (cardiac bed)
- • Low sodium diet
- • Strict bed rest
- • Ensure adequate nutrition
- • Correct hypoglycemia with 3-5ml/kg IV of Dextrose 10%
- Pharmaceutical treatment
- • Dopamine IV 5-10 microgram/kg/min, may increase to 20 microgram/kg/min Or
- • Dobutamine IV 2 to 20 microgram/kg/min

•Furosemide IV 2mg/kg/dose if adequate peripheral perfusion. Repeat the dose according to estimated fluid

overload up to 8mg/kg/day

- • Correct arrhythmia if present with Digoxin 0.04mg/kg/day in 3 divided doses (maintenance0.01mg/kg/day)
- • Monitor: Heart rate, Respiratory rate, BP, Urine output, Pulse Oxymetry for oxygen saturation

- **3.3. Pulmonary Oedema**

- 

- **Kerley B lines represent thickened connective tissue planes, for example due to edema of the septal lines of secondary lobules. They are most commonly due to pulmonary edema or lymphangitic carcinomatosis. Kerley B lines are horizontal, < 2 cm long and 1 mm thick, at periphery of lung and reaching the lung edge.**

- Definition: Pulmonary oedema is the accumulation of fluid in the
- alveoli due to an increase in pulmonary capillary venous pressure resulting from acute left ventricular failure.
- Causes
- - Heart not removing fluid from lung circulation properly (cardiogenic pulmonary edema)
- - A direct injury to the lung parenchyma
- Signs and Symptoms
- - Breathlessness/ respiratory distress
- - Sweating
- - Cyanosis (decreased oxygen saturation)
- - Frothy blood-tinged sputum
- - Ronchi, and crepitations/wheezers
- Investigations
- - Chest x-ray shows loss of distinct vascular margins, Kerley B lines, diffuse haziness of lung fields, pleural effusion
- - Blood Gas if possible
- - ECG
- - Echocardiography
- Management
- - Maintain patient in a semi sitting position
- - Oxygen by facial mask with reservoir bag if available

- - IV Furosemide 2mg/kg/dose, maximum 8mg/kg/day
- - Inotropic support with Dopamine or Dobutamine if signs of shock
- - Transfer to cardiologist for further management

- 3.4. Congenital Heart Diseases

- Definition: Congenital heart disease refers to a problem with the heart's structure and function due to abnormal heart developmentbefore birth. Often divided into two types, non-cyanotic and cyanotic(blue discoloration caused by a relative lack of oxygen).

General principles of management

• Initial stabilization: secure airway, adequate ventilation, circulatory support

• Correct metabolic acidosis, electrolyte derangements, hypoglycaemia;
prevent hypothermia.

• Empirical treatment with IV antibiotics.

• Early cardiology consultation.

• IV Prostaglandin E infusion if duct-dependent lesions suspected:

• Starting dose: 10 – 40 ng/kg/min; maintenance: 2 – 10 ng/kg/min.

• Adverse effects: apnoea, fever, hypotension.

• If unresponsive to IV prostaglandin E, consider:

• Transposition of great arteries, obstructed total anomalous pulmonary.
venous drainage.
• Blocked IV line.
• Non-cardiac diagnosis.

- • Arrangement to transfer to regional cardiac center once stabilized.
  - **3.4.1. Non Cyanotic Heart Diseases**

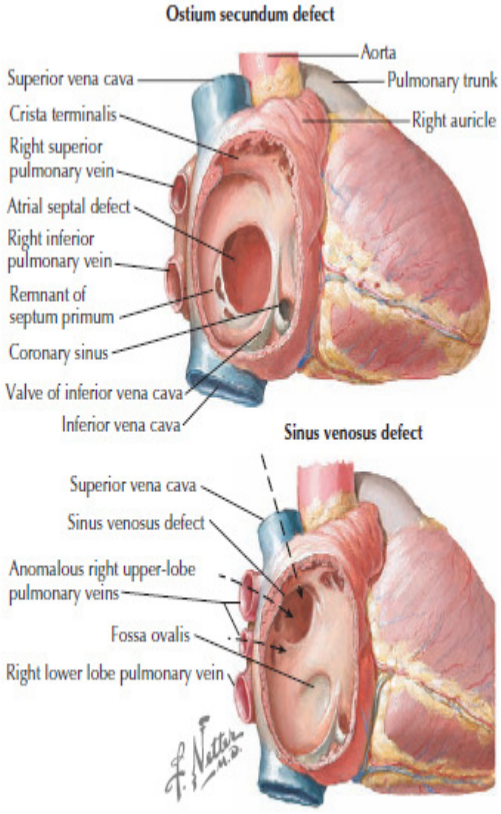


Figure 43-1 *Defects of the atrial septum.*

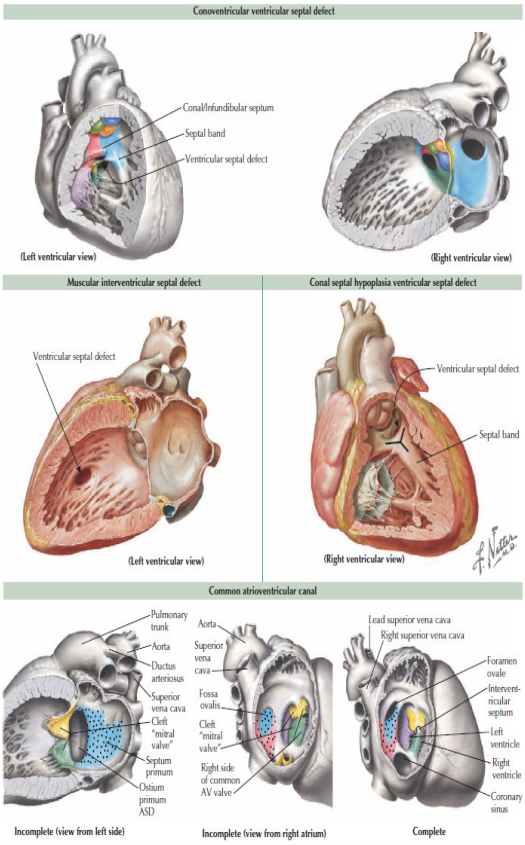
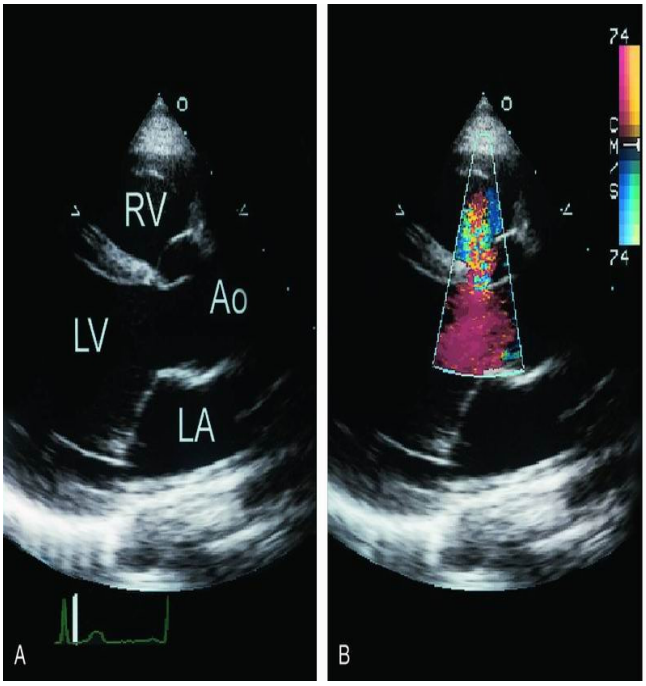
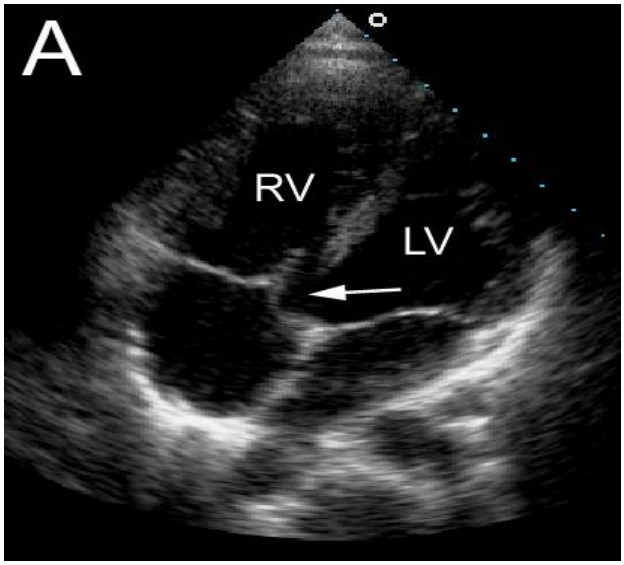


Figure 43-3 *Ventricular septal defect.*

- 

- Small ventricular septal defects may not be apparent on two-dimensional imaging **(A)**, but their presence can be confirmed using color Doppler imaging **(B)**. In this example, the septum appears intact, but medial angulation and the use of color Doppler imaging confirm the presence of a small defect. Ao, aorta; LA, left atrium; LV, left ventricle; RV, right ventricle.
- Common lesions
-



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-
- A trabecular ventricular septal defect (*arrow*) is shown. The presence of the defect is suggested on two-dimensional imaging
- - Ventricular Septal defect (VSD) most common congenital heart disease
- - Patent ductus arteriosus (PDA)
- - Atrio-ventricular septal defect (AVSD) or endocardial cushion defect (common in trisomy 21)
- Atrial septal defect (rarely causes heart failure)
- Coarctation of aorta
- Signs and Symptoms
- - Tachypnea, dyspnea

- - Tachycardia
- - Sweating
- - Feeding difficulties / failure to thrive
- - Recurrent chest symptoms
- - Hepatomegaly
- - Increased jugular venous pressure
- Complications
- - Failure to thrive
- - Infective Endocarditis
- Pulmonary vascular obstructive disease (pulmonary hypertension) which can lead to Eisenmenger syndrome
- Investigations
- - Chest X-Ray
- - ECG
- - Echocardiogram
- - Cardiac catheterization/angioscan in special cases
- Management
- Treatment depends on the specific condition. Some congenital heart diseases can be treated with medication alone, while others require one or more surgeries.
- - Lasix 2mg/kg/day
- - captopril 1-3mg/kg/day (start with 1mg/kg)
- - Increase calories in feeding
- - Iron if Hb less than 10g/dl (preferably reach 15g/dl)

- - Surgical repair generally before 1 year if possible

- **3.4.2. Cyanotic Heart Diseases**

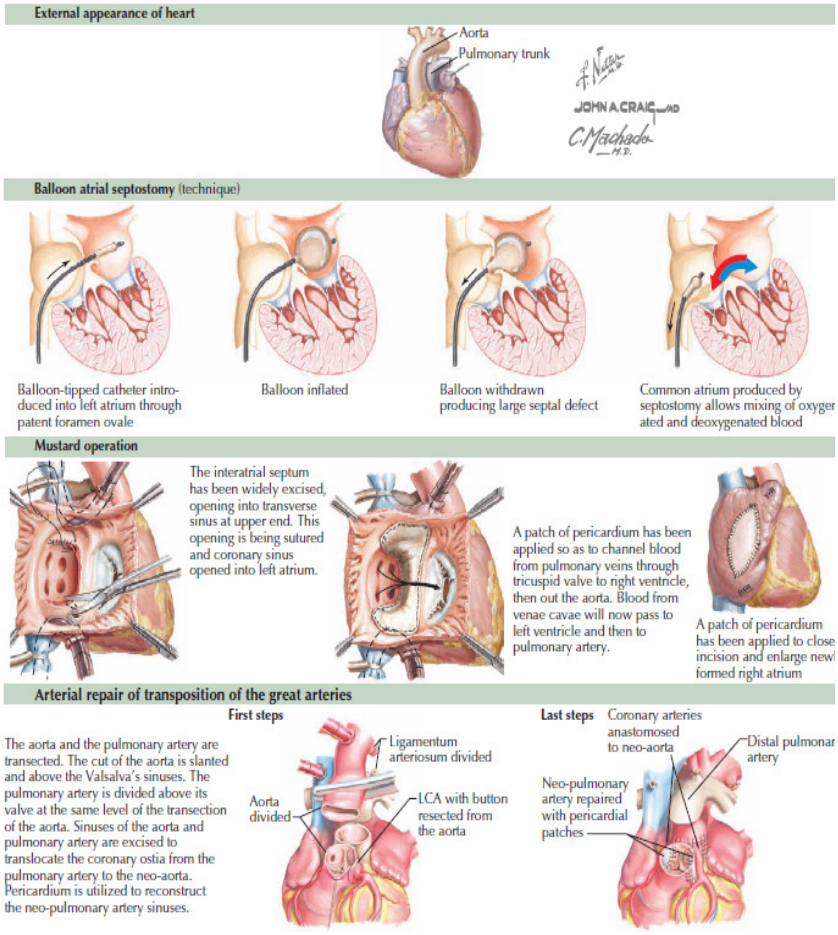
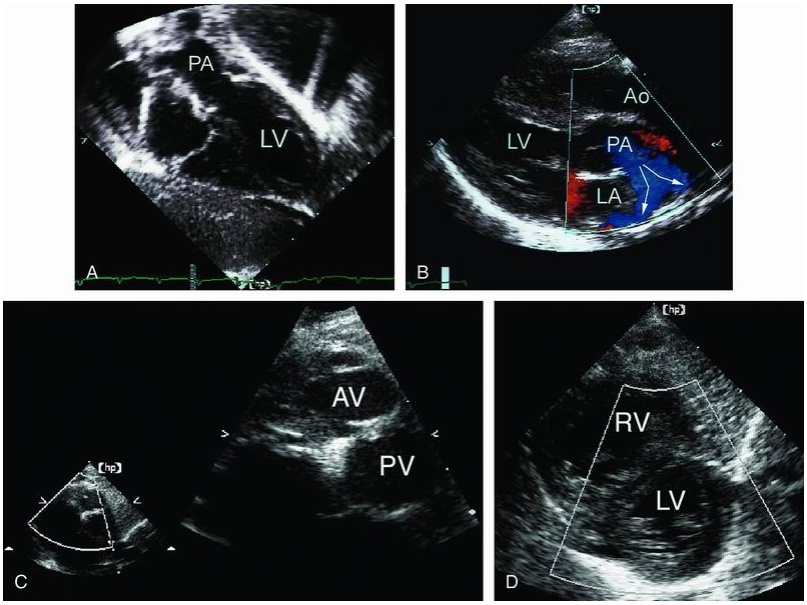


Figure 44-1 *Transposition of the great arteries.*

Fig. 6.12 Several cardiac abnormalities co-existing. Note enlarged heart.



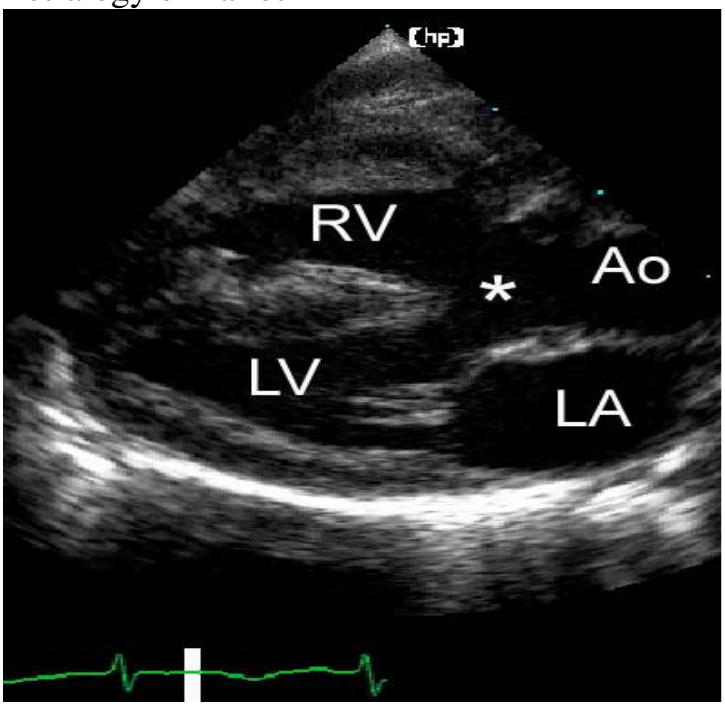
An example of D-transposition of the great arteries in an infant is shown. **A:** From the subcostal view, the pulmonary artery (*PA*) can again be seen to arise from the anatomic left ventricle (*LV*). **B:** By demonstrating

bifurcation of the great artery that arises from the posterior left ventricle, ventriculoarterial discordance is confirmed. **C:** A short-axis view at the base of the heart demonstrates the parallel course of the great arteries with an anterior aortic valve (*AV*). **D:** The right ventricle (*RV*) is seen anterior and rightward of the left ventricle. It is dilated and hypertrophied. Ao, aorta; LA, left atrium.

- Definition: Cyanotic heart disease is a heart defect, present at birth (congenital), that results in low blood oxygen levels (< 90 % even with oxygen).

Summary of The Clinical Approach to Cyanotic Newborns					
Cause	History, Signs	Chest X-ray	ABG	Hyperoxia test	Echocardiography
Cyanotic Heart Disease	No/mild Respiratory distress. Heart murmur.	Abnormal heart size and pulmonary vasculature	Low PCO_2	No rise in PO_2	Usually diagnostic
Primary Lung Disease	Respiratory distress	Abnormal lungs	Low PO_2 Hgh PCO_2	PO_2 >100mmHg	Normal
Persistent Pulmonary Hypertension	Suggestive history (MAS, asphyxia, sepsis)	Maybe abnormal (lungs)	Differential cyanosis	Inconclusive	Right to left shunt across PFO or PDA
Methemoglobinemia	Normal	Normal	Normal	PO_2 >100mmHg	Normal
MAS, meconium aspiration syndrome; PFO, patent foramen ovale; PDA, patent ductus arteriosus					

-
- Common lesions
- decreased flow to the lungs (does not cause heart failure)
- • Tetralogy of fallot
- • Pulmonary atresia
- - Increased flow to the lungs (does cause heart failure and failure to thrive):
- • Transposition of great vessels (TGA)
- • Truncus arteriosus

- • Single ventricle / Tricuspid atresia
- Tetralogy of Fallot
- 

- Long-axis image from a patient with tetralogy of Fallot demonstrates the overriding aorta (*Ao*) and a large subaortic ventricular septal defect (*asterisk*). Right ventricular hypertrophy is also present. LA, left atrium; LV, left ventricle; RV, right ventricle.
- Definition: Tetralogy of Fallot refers to a type of congenital heart defect comprising of:
- - Large ventricular septal defect

- - Narrowing of the pulmonary outflow tract (pulmonary stenosis)
- - Overriding aorta
- - Right ventricular hypertrophy
- Signs and Symptoms
- - Progressive cyanosis with pulmonary systolic murmur
- - digital clubbing occurs after long time
- - Hallmark: Paroxysmal hyper cyanotic attacks (blue spells) with the following manifestations
- • Hyperpnea and restlessness
- • Increased cyanosis
- • Gasping respiration
- • Syncope or convulsions
- •Spontaneous squatting position is frequent (in older children)
- • Heart murmur disappears
- Complications
- - delayed development/growth
- - Polycythemia
- - Hypercyanotic attack, sometimes associated with seizures and death

- Infective endocarditis
- Brain abscess
  - Investigations
  - - Chest x-ray
  - - Complete blood count (CBC)
  - - Echocardiogram

- - Electrocardiogram (ECG)
- Management
- - Avoid dehydration and stress
- - Propanolol 0.5-1mg/kg every 6 hours to prevent hypercyanotic attacks
- - Iron 5mg/kg /day to prevent microcytosis
- - Surgical repair, urgent as soon as spells begin
- - In case of Hypercyanotic attacks
- • Squatting position (hold the infant with the legs flexed on the abdomen)
- • Oxygen 6l/min with mask
- • Diazepam 0.3mg/kg IV or 0.5mg PR if convulsing
- • normal saline 10-20ml/kg/ 30 minutes
- • Sodium bicarbonate 8.5% 1ml/kg to correct acidosis
- • Morphine 0.1mg/kg IV if persistent attacks (but risk of
- respiratory depression)
- • Propranolol IV 0.1 – 0.2 mg/kg slowly then continue oral maintenance to relax the infundibular spasms
- Recommendations
- - All children with cyanotic heart diseases who come with diarrhea and vomiting should be admitted for closer observation.
- Furosemide is contra-indicated.

- - All new born babies with suspected cyanotic heart disease should be referred to a cardiologist/tertiary hospital immediately.
- - Common causes of heart failure in Neonates:

Clinical manifestations	Likely lesions
Very poor pulses	- Hypoplastic Left Ventricle Sy - Critical aortic stenosis
Poor femoral pulses	- Coarctation of aorta
Bounding pulses	- Patent ductus arterious (PDA - Troncus arteriosus - Severe anemia

-
  - **3.5. Acquired Heart Diseases**
  - **3.5.1. Acute Rheumatic fever**
-

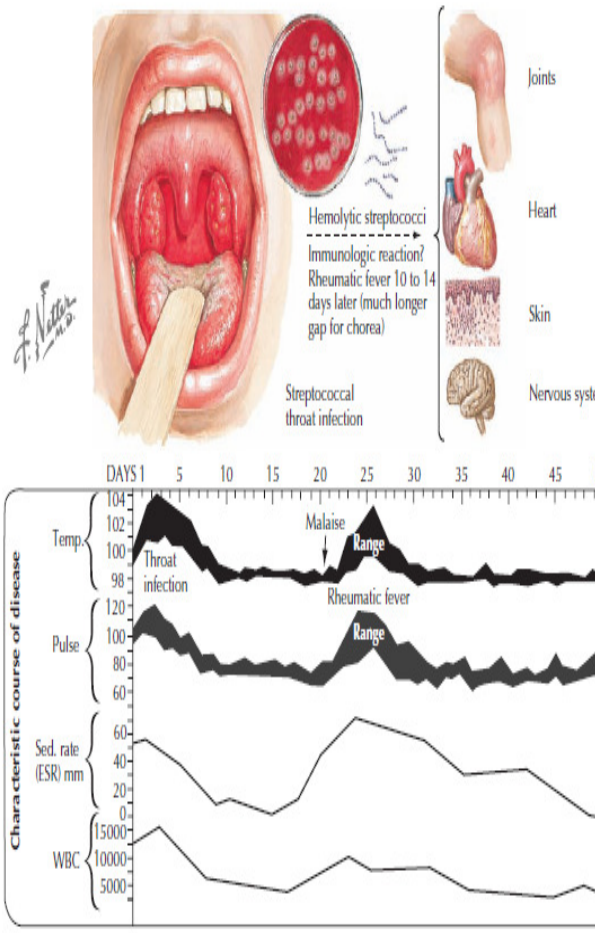


Figure 49-1 *Cardiac manifestations of acute rheumatic fever.*

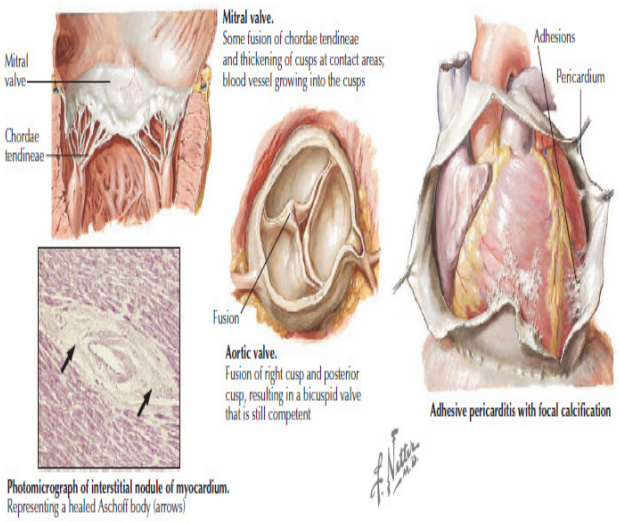


Figure 49-2 *Manifestations of acute rheumatic carditis.*

-
- Definition: This is an acute, systemic connective tissue disease in
- children related to an immune reaction to untreated group A Beta
- haemolytic streptococcus infection of the upper respiratory tract. The initial attack of acute rheumatic fever occurs in most cases between the ages of 3 and 15 years.
- Cause
- - Auto-immune disease
- Signs and Symptoms (Revised Jones Criteria)

Diagnostic criteria for Acute Rheumatic Fever		
Major Criteria	**Minor Criteria**	**Investigations**
Carditis	Fever (Temp > 38 °C)	FBC: anaemia, leucocytosis
Polyarthritis, aseptic monoarthritis or polyarthralgia	ESR > 30 mm/h or CRP > 30 mg/L	Elevated ESR and CRP
		Throat swab, ASOT
		Blood culture
Chorea	Prolonged PR interval	CXR, ECG.
Erythema marginatum		Echocardiogram
Subcutaneous nodules		

Making the Diagnosis:

- Initial episode of ARF:
  2 major criteria or 1 major + 2 minor criteria,
  \+ *evidence of a preceding group A streptococcal infection*
- Recurrent attack of ARF: (known past ARF or RHD)
  2 major criteria or 1 major + 2 minor criteria or 3 minor criteria,
  \+ *evidence of a preceding group A streptococcal infection*

Note:

1. Evidence of carditis: cardiomegaly, cardiac failure, pericarditis, tachycardia out of proportion to fever, pathological or changing murmurs.

2. Abbrevations: ARF, Acute Rheumatic Fever; RHD, Rheumatic Heart Disease

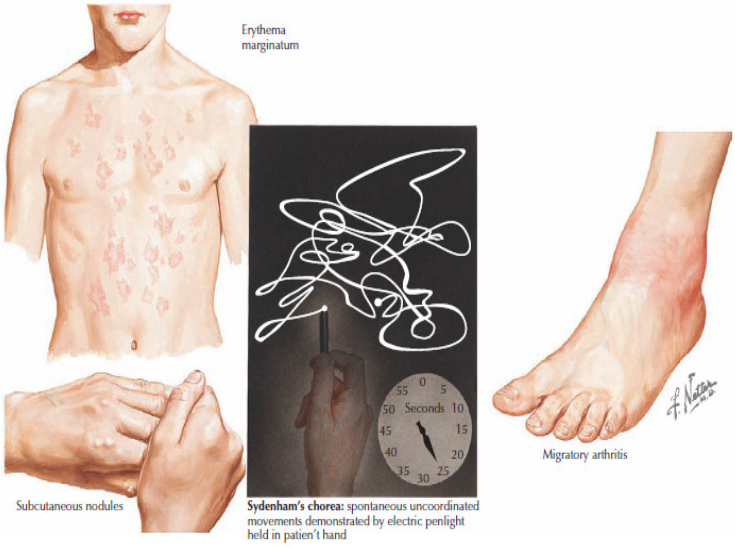


Figure 49-3 *Noncardiac manifestations of acute rheumatic fever.*

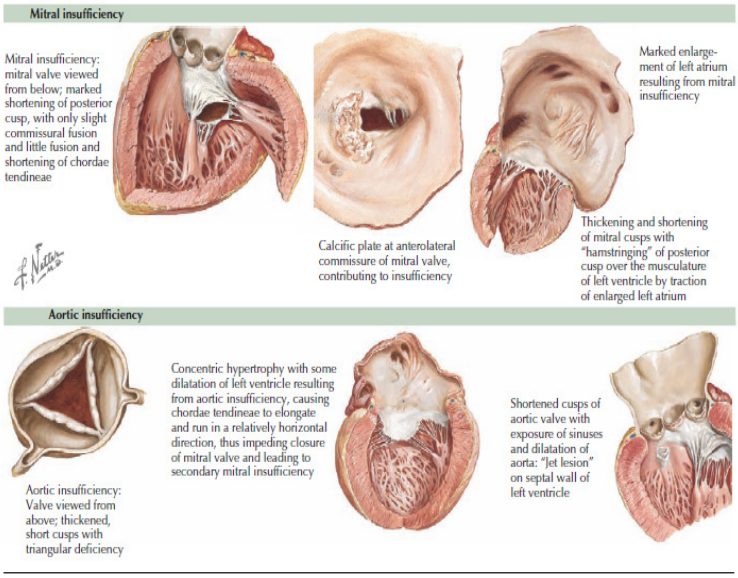


Figure 49-4 *Rheumatic heart disease: clinical presentation.*

Treatment

Aim to suppress inflammatory response so as to minimize cardiac damage,
provide symptomatic relief and eradicate pharyngeal streptococcal infection

• Bed rest. Restrict activity until acute phase reactants return to normal.

• Anti-streptococcal therapy:

• IV C. Penicillin 50 000U/kg/dose 6H
or Oral Penicillin V 250 mg 6H (<30kg), 500 mg 6H (>30kg) for 10 days

• Oral Erythromycin for 10 days if allergic to penicillin.

• Anti-inflammatory therapy

• *mild / no carditis:*
Oral Aspirin 80-100 mg/kg/day in 4 doses for 2-4 weeks, tapering over 4 weeks.

• *pericarditis, or moderate to severe carditis:*
Oral Prednisolone 2 mg/kg/day in 2 divided doses for 2 - 4 weeks,taper with addition of aspirin as above.

• anti-failure medications

• Diuretics, ACE inhibitors, digoxin (to be used with caution).

Important:

- *Consider early referral to a Paediatric cardiologist if heart failure persists or worsens during the acute phase despite aggressive medical therapy. Surgery may be indicated.*

Secondary Prophylaxis of Rheumatic Fever

- IM Benzathine Penicillin 0.6 mega units (<30 kg)
  or 1.2 mega units (>30 kg) every 3 to 4 weeks.
- Oral Penicillin V 250 mg twice daily.
- Oral Erythromycin 250 mg twice daily if allergic to Penicillin.

Duration of prophylaxis

- Until age 21 years or 5 years after last attack of ARF whichever was longer
- Lifelong for patients with carditis and valvular involvement.

- Chorea

→ Most mild-moderate cases do not need medication

→ Provide calm and supportive environment (prevent accidental self-harm)

→ For severe cases: carbamazepine per os

- <6 years: 10-20mg/kg/day divided in 3 doses
- 6-12 years: 400-800mg/day divided in 3 doses
- >12 years: 200mg x 2/day

OR

→ Valproic acid 20-30mg/kg/day divided in 2 doses; duration: 2 weeks

- Carditis

→ Bed rest if in cardiac failure

→ Anti-failure medication as above

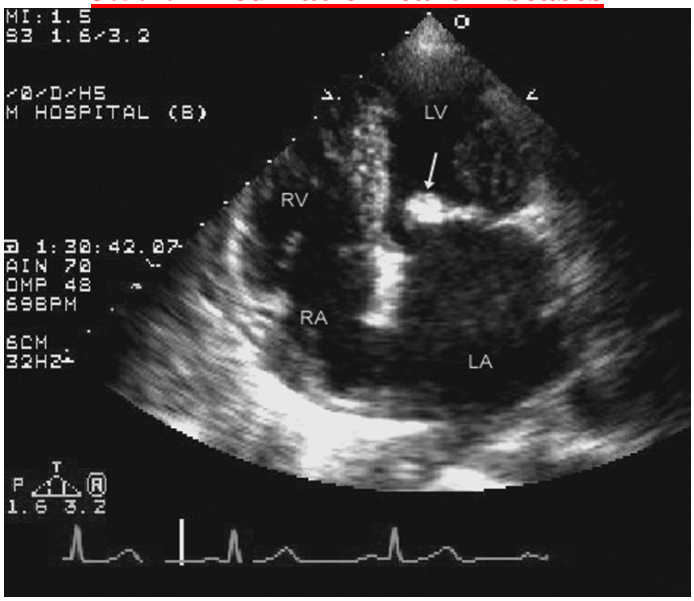
→ Anti-coagulation medication if atrial fibrillation is present

- Recommended duration of Secondary Prophylaxis

Disease Classification	Duration of Secondary Prophylaxis
ARF with No proven carditis	- Minimum of 5 years after last ARF, Until age 18 years (*whichever is lon*
Mild-moderate RHD (or healed carditis)	- Minimum 10 years after last ARF, o - Until age 25 years (*whichever is lon*
Severe RHD and following Cardiac Surgery for RHD	- Continue medication for life

-
-

- **3.5.2. Rheumatic Heart Diseases**



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- Apical four-chamber view recorded in a patient with rheumatic mitral stenosis. Note the marked dilation of the left atrium (*LA*). In this example, there is substantial but focal calcification of the anterior mitral valve leaflet (*arrow*). Note also the relatively restricted motion of both leaflets along their full length. LV, left ventricle; RA, right atrium; RV, right ventricle.

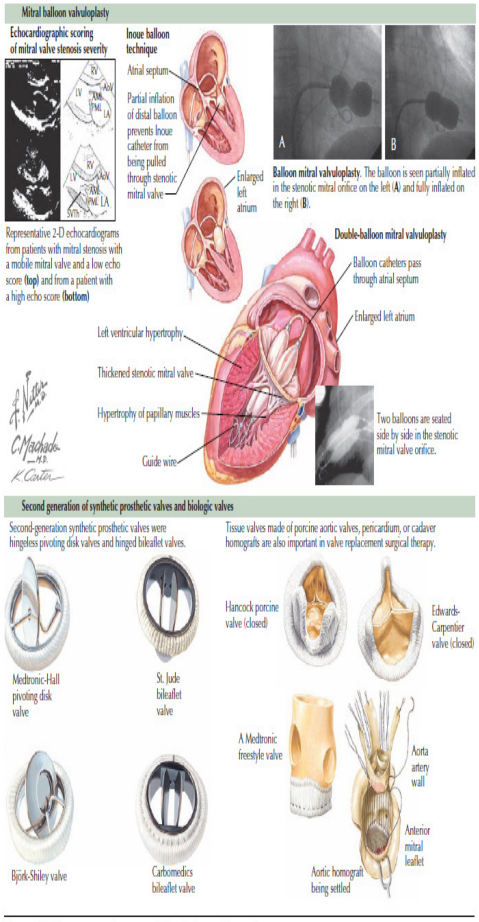


Figure 49-5 *Management and treatment of chronic rheumatic heart disease.*

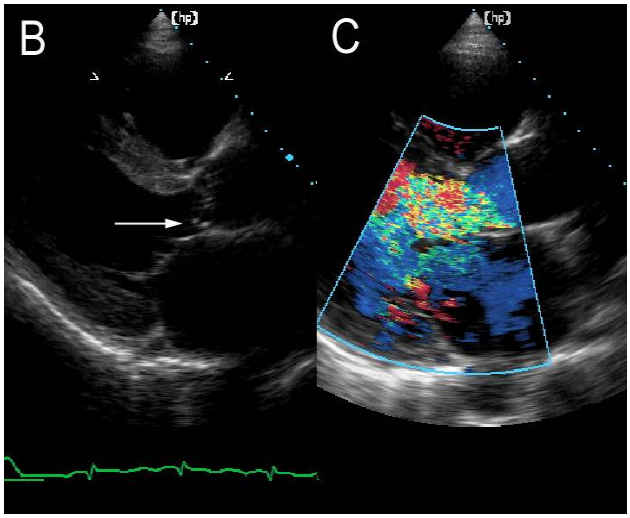
- Definition: It is an inflammatory damage of the heart valves, as a complication of acute rheumatic fever. The mitral valve is the most commonly involved valve, although any valve may be affected.

- Types of valvular lesions
- - Mitral regurgitation/stenosis
- - Aortic regurgitation/stenosis
- - Tricuspid regurgitation
- - Mixed regurgitation and stenosis
- - Multivalvular heart diseases
- Signs and Symptoms
- - May be asymptomatic when minor lesions
- - Heart murmurs over affected valve
- Complications
- - Congestive cardiac failure with pulmonary oedema
- - Bacterial endocarditis
- Investigations
- - Chest x-ray
- - ECG
- - Echocardiography
- Management
- - Treat underlying complication, e.g., heart failure, pulmonary oedema
- - Continue prophylaxis against recurrent rheumatic fever
- - Ensure oral hygiene
- - Endocarditis prophylaxis if dental procedures, urinary tract instrumentation, and GIT manipulations
- • Procedure done above the diaphragm

- → Amoxicillin 50mg/kg (Max 2gr) 1 hour before the procedur
- Or→ Erythromycin 50mg/kg (max 1.5gr) – if allergic to Penicillins
- • Below the diaphragm
- → Ampicillin 50mg/kg IV or IM (max 2gr) with
- Gentamycine, 2mg/kg (max 120mg) 30minutes before the procedure
- Then
- → Amoxycillin per os 25mg/kg (max1gr) 6 hours after the procedure
- - Ensure good follow up by cardiologist

- 3.5.3. Infective Endocarditis (IE)

Figure 17.21 Widespread infected emboli and infarcts in a child with bacterial endocarditis. The tip of the third toe is gangrenous.



- A small aortic valve vegetation (*arrow*) is shown during diastole **(A)** and systole **(B)**. **C:** Color Doppler demonstrates severe aortic regurgitation. Ao, aorta; LA, left atrium; LV, left ventricle; RV, right ventricle.

- Definition: Infection of the endothelial surface of the heart. Suspectc infective endocarditis in all children with persistent fever and underlying heart disease.
- Causes/predisposing factors
- - Rheumatic valvular disease
- - Congenital heart disease
- Signs and Symptoms
- - Persistent low grade fever without an obvious underlying cause

- - Fatigue, joint pain, new murmurs, clubbing, splenomegaly and haematuria
- DUKE CRITERIA IN CHILDREN

Modified Duke Criteria for the Diagnosis of Infective Endocarditis	
Major Criteria	**Minor Criteria**
• Blood culture positive: *Typical microorganisms from two separate blood cultures:*	• Predisposing heart condition, prior heart surgery, indwelling catheter
	• Fever, temperature > 38°C
• *Viridans streptococci*	• Vascular phenomena:
• *Streptococcus bovis*	• Major arterial emboli
• *HACEK group*[1]	• Septic pulmonary infarcts
• *Staphylococcus aureus*	• Mycotic aneurysm
• *Community-acquired enterococci*	• Intracranial hemorrhage,
	• Conjunctival hemorrhages
• Evidence of endocardial involvement on echocardiogram	• Janeway's lesions
	• Immunologic phenomena:
	• Gomerulonephritis
Footnote: *1, Fastidious gram negative bacteria from Haemophilus spp, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens and Kingella kingae*	• Osler's nodes
	• Roth's spots
	• Positive Rheumatoid factor
	• Microbiological evidence:
	• Positive blood culture not meeting major criterion

Definition of Infective Endocarditis According to the Modified Duke Criteria		
Definite IE	**Possible IE**	**Rejected IE**
Pathological criteria • Microorganisms by • Culture • Histological examination of vegetation or intra-cardiac abscess specimen. • pathological lesions with active endocarditis. *Clinical criteria* • 2 major or • 1 major + 3 minor or • 5 minor	• 1 major + 1 minor criteria OR • 3 minor	• Firm alternative diagnosis • Resolution of symptoms with antibiotic therapy < 4 days. • No pathological evidence of IE at surgery or autopsy. • Not meet criteria for possible IE.
Footnote: IE, Infective Endocarditis		

Investigations

- Blood culture
- C- Reactive protein/ESR
- Full blood count
- Urine FEME
- Chest X-ray
- Echocardiography

Management

- Ensure 3 blood cultures taken before antibiotic therapy.
- Do not wait for echocardiography.
- Use empirical antibiotics, until culture results available (see Table on faci

Antibiotic choices for Infective endocarditis in Children (Adapted from Malaysian CPG on antibiotic usage)		
Indication	**Preferred Regime**	**Alternative Regime**
Empirical Therapy For Infective Endocarditis	IV Penicillin G 200,000 U/kg/day in 4-6 div doses x 4wks AND IV/IM Gentamicin 3 mg/kg/day in 3 div doses x 2wks	IV Vancomycin 30 mg/kg/day in 2 div doses x 4–6wks AND IV/IM Gentamicin 3 mg/kg/day in 3 div doses x 2wks
Streptococcus viridans endocarditis	IV Vancomycin 30 mg/kg/day in 2 div doses x 4–6 wks AND IV/IM Gentamicin 3 mg/kg/day in 3 div doses x 2wks	IV Vancomycin 30 mg/kg/day in 2 div doses x 4–6wks AND IV/IM Gentamicin 3 mg/kg/day in 3 div doses x 2wks
Enterococcus endocarditis	IV Penicillin G 300,000 U/kg/day in 4-6 div doses x 4 - 6wks AND IV Gentamicin 3 mg/kg/day in 3 div doses x 4 - 6 wks	
Methicillin sensitive Staphylococcus endocarditis	IV Cloxacillin 200mg/kg/day in 4-6 div doses x 6wks +/- IV/IM Gentamicin 3mg/kg/day in 3 div doses x 3-5 days	
Penicillin allergy Methicillin Resistance	IV Cefazolin 100mg/kg/day in 3 div doses x 6 wks IV Vancomycin 40 mg/kg/day in 2-4 div doses x 6wks	IV Vancomycin 40 mg/kg/day in 2 div doses x 4–6wks
Culture- Negative Endocarditis	IV Ampicillin-Sulbactam 300mg/kg/day in 4-6 div doses x 4-6 wks AND IV Gentamicin 3mg/kg/day in 3 div doses x 4-6 wks	IV Vancomycin 40 mg/kg/day in 2 div doses x 4–6wks AND IV/IM Gentamicin 3 mg/kg/day in 3 div doses x 4-6wks AND IV Ciprofloxacin 20-30 mg/kg/day in 2 div doses x4-6wks
Fungal Endocarditis Candida spp or Aspergillosis	IV Amphotericin B > 6 weeks AND Valve replacement surgery AND Long-term (lifelong) therapy with Oral azole	

Guidelines on Infective Endocarditis (IE) prophylaxis	
IE prophylaxis Recommended	**IE prophylaxis Not Recommended**
High-risk category • Prosthetic cardiac valves. • Previous bacterial endocarditis. • Complex cyanotic congenital heart disease. • Surgical systemic pulmonary shunts or conduits.	*Negligible-risk category* • Isolated secundum ASD. • Repaired ASD, VSD, PDA (> 6 mths) • Mitral valve prolapse without regurgitation. • Functional, or innocent heart murmurs. • Previous Kawasaki disease without valvar dysfunction. • Previous rheumatic fever without valvar dysfunction. • Cardiac pacemakers and implanted defibrillators.
Moderate-risk category • Other congenital cardiac defects (other than high/low risk category) • Acquired valvar dysfunction. (e.g. rheumatic heart disease) • Hypertrophic cardiomyopathy. • Mitral valve prolapse with regurgitation.	
Common procedures that require IE Prophylaxis	
Oral, dental procedures • Extractions, periodontal procedures. • Placement of orthodontic bands (but not brackets). • Intraligamentary local anaesthetic injections. • Prophylactic cleaning of teeth. *Respiratory procedures* • Tonsillectomy or adenoidectomy. • Surgical operations involving respiratory mucosa. • Rigid bronchoscopy. • Flexible bronchoscopy with biopsy.	*Gastrointestinal procedures* • Sclerotherapy for esophageal varices. • Oesophageal stricture dilatation. • Endoscopic retrograde cholangiography biliary tract surgery. • Surgical operations involving intestinal mucosa. *Genitourinary procedures* • Cystoscopy. • Urethral dilation.
Antibiotic guidelines for IE prophylaxis	
Endocarditis Prophylactic Regimens for Dental, Oral, Respiratory Tract and Esophageal Procedures	
Standard general prophylaxis	Penicillin allergy (Either one of below):
• Oral Amoxicillin 50 mg/kg (max 2 Gm), one hour before procedure *OR* • IV/IM Ampicillin 50 mg/kg (max 2 Gm)	• Oral Clindamycin 20 mg/kg (max 600 mg) • Oral Cephalexin 50 mg/kg (max 2 Gm) • Oral Azithromycin/clarithromycin 50 mg/kg (max 500 mg) • Oral Erythromycin 20 mg/kg (max 3 Gm) • IV Clindamycin 20 mg/kg (max 600 mg)
Note: Give oral therapy 1 hour before procedure; IV therapy 30 mins before procedure.	

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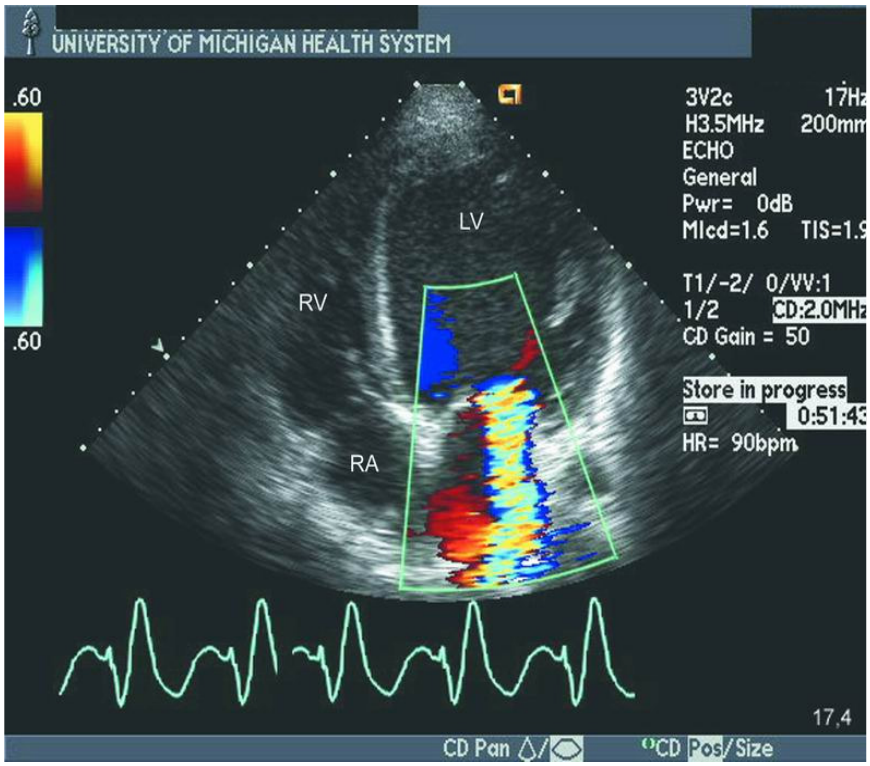
3.6. Cardiomyopathies

3.6.1. Dilated Cardiomyopathy



Apical four-chamber view recorded in a patient with a dilated cardiomyopathy and functional mitral regurgitation that is the result of lateral displacement of the papillary muscles. Note the mitral regurgitation jet, with the color flow Doppler signal filling approximately 40% of the left atrial area. LV, left ventricle; RA, right atrium; RV, right ventricle.

Definition: dilated cardiomyopathy refers to a group of conditions of diverse etiology in which both ventricles are dilated with reduced contractility.

Classification

Classification based on the predominant structural and functional abnormalities

- dilated Cardiomyopathy: primarily systolic dysfunction
- Hypertrophic Cardiomyopathy: primarily diastolic dysfunction
- Restrictive Cardiomyopathy: primarily diastolic but often combined with systolic dysfunction

Causes

- Infections (e.g. Viral+++, Rickettsia, Chagas disease)
- Neuromuscular disorders (e.g. duchenne dystrophy, Becker dystrophy)
- Endocrine, metabolic and nutritional (e.g. hyperthyroidism, Fatty acid oxidation disorders, beriberi, kwashiorkor)
- diseases of coronary arteries (e.g. Kawasaki, Aberrant Left Coronary Artery - ALCAPA)
- Autoimmune diseases (e.g. Rheumatic carditis, juvenile rheumatoid arthritis, systemic lupus erythematosus, dermatomyositis, systemic lupus erythematosus)
- drugs toxicity (e.g. doxorubicin,cyclophosphamide, IPECA)
- Hematologic diseases (e.g. anemia, Sickle cell anemia, hypereosinophilic syndrome Löffler syndrome)

Signs and Symptoms (See signs of congestive heart failure)

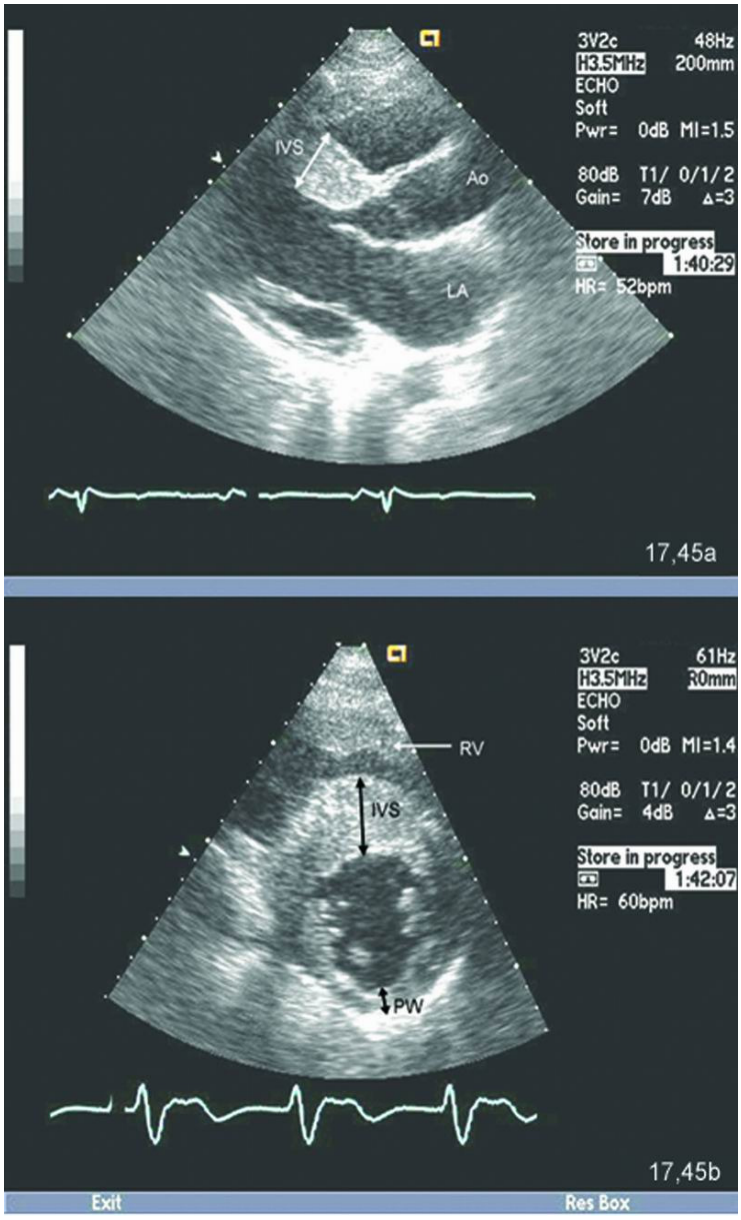
Diagnosis

- ECG: proeminent P wave, LV or RV hypertrophy, nonspecific T-wave abnormalities
- Chest X-ray: cardiomegaly, pulmonary edema
- Echocardiogram: confirm diagnosis and shows LA and LV dilation, poor contractility
- FBC, Urea and creatinine, Electrolytes (Na, K)
- Myocardial biopsy, PCR

Management

- Treatment: (Refer to principles and medication of congestive heart failure)

3.6.2. Hypertrophic Cardiomyopathy


3V2c 48Hz
H3.5MHz 200mm
ECHO
Soft
Pwr= 0dB MI=1.5
80dB T1/ 0/1/2
Gain= 7dB Δ=3
Store in progress
1:40:29
HR= 52bpm
IVS
Ao
LA
17,45a
3V2c 61Hz
H3.5MHz R0mm
ECHO
Soft
Pwr= 0dB MI=1.4
80dB T1/ 0/1/2
Gain= 4dB Δ=3
Store in progress
1:42:07
HR= 60bpm
RV
IVS
PW
17,45b
Exit
Res Box

Parasternal long-axis **(A)** and short-axis **(B)** views recorded in a patient with classic hypertrophic cardiomyopathy. In both the long-axis and short-axis views, note the marked thickening of the interventricular septum and the normal thickness of the posterior wall (*PW*). In the short axis view, note that there is a spectrum of hypertrophy of the left ventricle, with maximum hypertrophy in the septum, no hypertrophy of the true posterior wall, and intermediate hypertrophy of the lateral and true inferior wall. Ao, aorta; IVS, interventricular septum; LA, left atrium; RV, right ventricle.

Causes

- Left ventricle obstruction (Coartation of aorta, hypertension, aortic stenosis)
- Secondary (infants of diabetic mothers, corticosteroids in premature infants)
- Metabolic (Glycogen storage disease type II (Pompe disease)
- Familiar hypertrophic cardiomyopathy
- Syndroms (Beckwith - Wiedman syndrom, Friedereich, ataxia)

Signs and Symptoms

- Weakness
- Fatigue
- dyspnea on effort
- Palpitations
- Angina pectoris

- dizziness and syncope
- Increased risk of sudden death

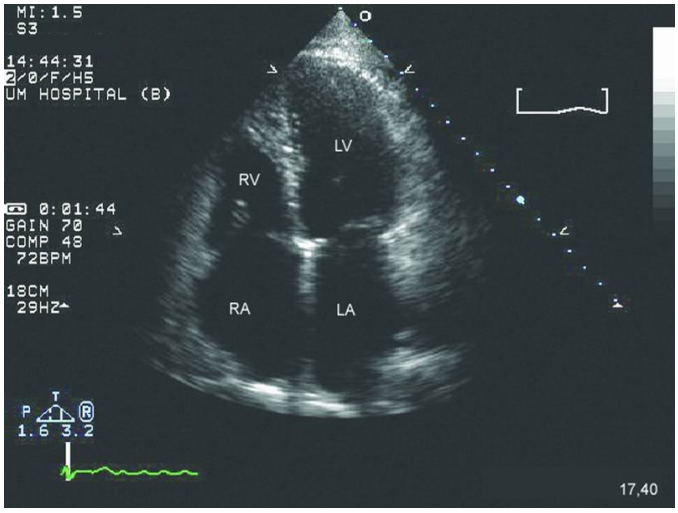
Diagnosis

- ECG: LV hypertrophy
- Chest X-ray: Mild cardiomegaly
- Echocardiogram: LV hypertrophy, ventricular outflow tract gradient
- doppler flow studies may demonstrate diastolic dysfunction before the development of hypertrophy

Management

- Prohibit competitive sports and strenuous physical activities
- Propranolol 0.5 -1mg/kg/day devised in 3 doses or Atenolol
- Implantable cardioverter-defibrillator if documented arrhythmias or a history of unexplained syncope
- Open heart surgery for septal myotomy: rarely indicated

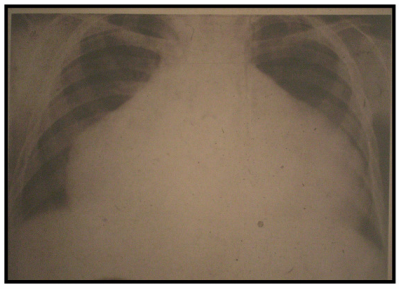
3.6.3. Restrictive Cardiomyopathy

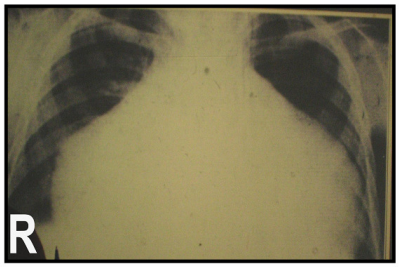


- Apical four-chamber view recorded in a patient with an idiopathic restrictive cardiomyopathy. This was a 70-year-old patient with refractory congestive heart failure and atrial fibrillation. Note the marked biatrial enlargement and normal right and left ventricular sizes. Left ventricular systolic function was normal. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.
- Definition: Restrictive cardiomyopathy refers to a group of disorders in which the heart chambers are unable to properly fill with blood because of stiffness in the heart muscle. Its prognosis is poor, and clinical deterioration can be rapid.
- Causes

- - Idiopathic, Systemic disease (scleroderma, amyloidosis, or sarcoidosis)
- - Mucopolysaccharidosis
- - Hypereosinophilic syndrome; malignancies
- - Radiation therapy
- - Isolated non compaction of the left ventricular myocardium
- Signs and Symptoms
- - dyspnea
- - Edema and ascites
- - Hepatomegaly with increased venous pressure
- - Pulmonary congestion
- Complications
- - Arrhythmias
- - Mitral regurgitation
- - Progressive heart failure
- - Tricuspid regurgitation
- Investigations
- - ECG: Prominent P waves, ST segment depression, T-wave inversion
- - Chest X-ray: mild to moderate cardiomegaly
- - Echocardiogram: markedly enlarged atria and small to normal-sized ventricles with often preserved systolic function but highly abnormal diastolic function
- Management
- - Lasix 2mg/kg divided in 2 doses

- - Aldactone 1-2mg/kg devised in 2 doses
- - Antiarrhythmic agents / biventricular pacing are used as required
- - Aspirin or Warfarin in case of non compaction LV with an increased risk of mural thrombosis and stroke
- - Cardiac transplantation where possible and indicated
- **3.7. Pericarditis/Pericardial Effusion**





Definition: Pericarditis is the inflammation of the pericardium.

- Pericardial effusion is the abnormal build-up of excess fluid that develops between the pericardium, the lining of the heart, and the heart itself.
- Causes
- - Infection such as viral, bacterial (tuberculosis)
- - Inflammatory disorders, such as lupus

- - Cancer that has spread (metastasized) to the pericardium
- - Kidney failure with excessive blood levels of nitrogen
- - Heart surgery (postpericardectomy syndrome).
- Signs and Symptoms
- - Pericardial tamponade
- - Chest pressure or pain and signs of congestive heart failure which can sometimes lead to shock.

> **Note:** *Many patients with pericardial effusion have no symptoms. The condition is often discovered on a chest x-ray or echocardiogram that was performed for another reason.*

-
- Investigations
- - ECG
- • Small complexes tachycardia, diffuse T wave changes
- - Chest X-ray: “water bottle” heart, or triangular heart with smoothed out borders
- - Echocardiogram
- - Tuberculin skin test
- - diagnostic pericardiocentesis
- • In all patients with suspected bacterial or neoplastic pericarditis and patients whom diagnosis is not readily obtained

- - Cell count and differential, culture, gram stain, PCR
- Management
- Non-pharmacological
- • Semi-sitting position if tamponnade suspected
- • Pericardiocentesis
- → preferably under ultrasound guidance
- → Performed by an experienced person
- → Indicated in children with symptomatic pericardial effusion
- Pharmacological
- • If hypotensive, rapidly administer intravenous fluids 20ml/kg of Normal saline over 30min to 1 hour
- • If suspected TB pericarditis: standard anti TB treatment + steroids
- • In case of purulent pericarditis: cloxacillin, IV 50 mg/kg/dose 6 hourly for 3 – 4 weeks + ceftriaxone, IV, 100 mg/kg as a single daily dose, to adapt according to culture results.
- • Treat heart Failure (See section on heart failure)
- Recommendation
- - All patients with pericardial effusion should be referred to a cardiologist

- 3.8. Hypertension in children

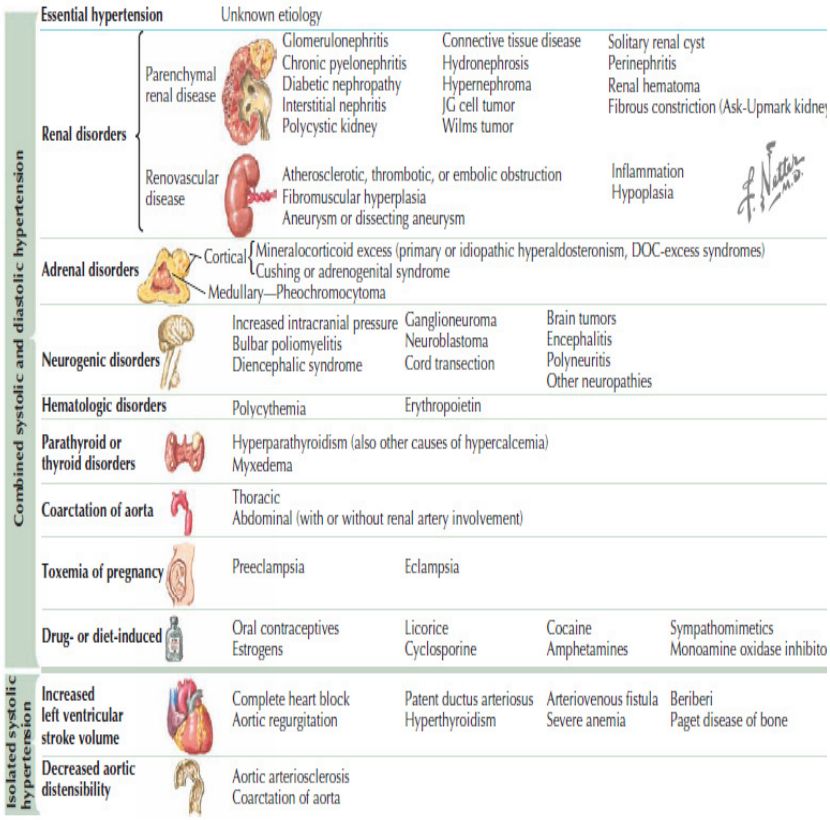


Figure 47-1 *Causes of hypertension.*

- Definition: Hypertension is defined as systolic and/or diastolic blood
- pressure ≥ the 95th percentile for gender, age and height percentile on at least three consecutive occasions.

- A sustained blood pressure of > 115/80 is abnormal in children between 6 weeks and 6 years of age.
- Causes
  - **Renal**
    - Renal parenchymal disease
    - Renovascular, e.g. renal artery stenosis
    - Polycystic kidney disease (ARPKD and ADPKD)
    - Renal tumours
  - **Coarctation of the aorta**
  - **Catecholamine excess**
    - Phaeochromocytoma
    - Neuroblastoma
  - **Endocrine**
    - Congenital adrenal hyperplasia
    - Cushing syndrome or corticosteroid therapy
    - Hyperthyroidism
  - **Essential hypertension**
    - A diagnosis of exclusion.
-
- Signs and Symptoms

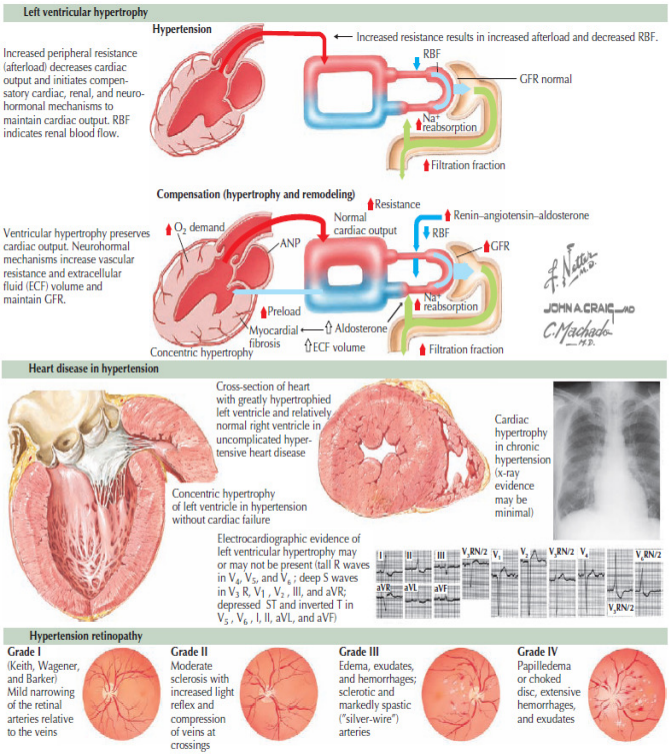


Figure 47-2 *Clinical presentation of hypertension.*

-
- - Headache
- - Convulsions, coma and visual symptoms
- - Oedema, haematuria, proteinuria
- - Acute heart failure and pulmonary oedema
- - Some children may be asymptomatic
- Blood pressure in children correlates with body size and age

Age of child	95th Percentile of Systolic and Diastolic Blood Pressure	
	First 12 hours	First week
newborn prem	65/45 mmHg	80/50 mmHg
newborn fullterm	80/50 mmHg	100/70 mmHg
	Systolic mmHg	Diastolic mmHg
6 weeks-6 Years	115	80
8 years	120	82
9 years	125	84
10 years	130	86
12 years	135	88
14 years	140	90

- 95th Percentile of systolic and diastolic BP correlated with Height

Height cm	Systolic mmHg	Diastolic mmHg
100	114	70
110	116	72
120	118	74
130	120	74
140	125	75
150	130	75
160	135 (131)	77
170	140 (133)	80
180	145 (135)	83

-
- Investigations
- - Urea, creatinine, electrolytes (Na+, K+)
- - Fundoscopy
- - ECG
- - Echocardiogram
- - Abdominal ultrasound (focused on kidneys)
- - Others according to the suspected etiology
- Management

- Acute hypertension (hypertension of sudden onset)
- Non-pharmacological
- • Admit patient to paediatric high dependence care unit
- • Monitor BP every 10 minutes until stable – thereafter every 30 minutes for 24 hours
- • Insert two peripheral intravenous drips
- • Rest on cardiac bed
- • Control fluid intake and output (restriction)
- • Restrict dietary sodium
- Pharmacological
- • do not combine drugs of the same class
- • Furosemide, IV, 1–2 mg/kg as a bolus slowly over 5 minutes, Increase up to 8 mg/kg/day
- • nifedipine 0.25-0.5mg/kg (max: 10mg) sublingual OR
- Amlodipine, oral, 0.2 mg/kg/dose. May be repeated 6 hours later, thereafter every 12 hours
- • Refer the patient to a specialist when the patient is stable
- Recommendations
- - For acute or chronic hypertension Blood Pressure needs to be lowered cautiously
- - Aim to reduce the SBP slowly over the next 24 - 48 hours,do not decrease BP to < 95th percentile in first 24 hours

- - Advise a change in lifestyle
- - Institute and monitor a weight reduction program for obese individuals
- - Regular aerobic exercise is recommended in essential hypertension
- - dietary advice
- - Limit salt and saturated fat intake
- - Increase dietary fiber intake
- Chronic Hypertension
- Non-pharmacological management
- • Introduce physical activity, diet management and weight reduction, if obese
- • Advise against smoking in teenagers
- • Follow up to monitor Blood Pressure and educate patient on hypertension
- • If Blood Pressure decreases, continue with non-drug management and follow up
- • If BP is increasing progressively, reinvestigate to exclude secondary causes or refer to the specialist
- •If BP is stable but persistently > 95th percentile and secondary causes have been excluded, start drug treatment after failed non-drug management for 6 months
- • Consider earlier initiation of drug treatment if positive family history for cardiovascular disease, essential hypertension or diabetes mellitus

- Pharmacological management
- Recommended medication and dosage for patients with chronic Hypertension

Recommended Hypertension medication for patients with Renal Failure

For CKD 1-3 (GFR>=30, creatinine <2x normal value for age	
First- line drug	*Lisinopril*
Second -line drug	*Hydrochlorothiazide*
Third- line drug	*Amlodipine*
Forth- line drug	*Atenolol* (use half of normal recommended dose)
For CKD 4 or 5 (GFR < 30, creatinine >=2x normal value for age	
First-line drug	*Furosemide*
Second-line drug	*Amlodipine*
Third-line drug	*Atenolol* (use half of normal recommended dose)

-
- Recommendations
- - All patients with hypertension and persistent proteinuria should be treated with an ACE inhibitor
- - Always exclude bilateral renal artery stenosis before treating with an ACE inhibitor
- - Renal function must be monitored when an ACE inhibitor is prescribed because it may cause a decline in GFR resulting in deterioration of renal function and hyperkalaemia

- - Patients with hypertension due to a neuro-secretory tumour
- (phaeochromocytoma or neuroblastoma) should receive an a-blocker either as single drug or in combination with ß-adrenergic blocker
- For patients with persistent hypertension despite the use of first line drugs, a second/third drug should be added
- Specific classes of antihypertensive drugs should be used according to the underlying pathogenesis or illness
- - For patients with a predominant fluid overload: use diuretics with/without ß-blocker

3.9. Cardiac Arrhythmias in children

- Definition: Heart rate that is abnormally slow or fast for age or irregular.
- There are three types of arrhythmias in children
- - Heart block
- - Ventricular arrhythmias
- - Paroxysmal atrial tachycardia

Type of Arrhythmia	Causes	Signs and symptom
Heart block: A delay or complete block of the electrical impulse as it travels from the sinus node to the ventricles	- Idiopathic and familial - Electrolyte disturbances (hyperkalaemia), - digoxin toxicity - Congenital heart disease, particularly transposition of the great arteries, and especially after surgery - Myocarditis - Post infective, for example in endocardial fibroelastosis or rheumatic fever	- Chest pressure or pain - Fainting, also known as syncopy, or near-syncope - Fatigue - Lightheadedness or dizziness - Palpitations, which can be skipping, fluttering or pounding in the chest - Shortness of breath
Ventricular arrhythmias: A rapid heart rate, usually with a regular rhythm, originating from above the ventricles	- Heart attack - Cardiomyopathy - Heart failure - Heart surgery - Myocarditis - Valvular heart disease	- May be asymptomatic - Chest disconfort (angina) - Fainting (syncope) - Light-headedness or dizziness - Sensation of feeling the heart beat (palpitations) - Shortness of breath - Absent pulse - Loss of consciousness - Normal or low blood pressure - Rapid pulse
Paroxysmal atrial tachycardia: A rapid heart rate, usually with a regular rhythm, originating from above the ventricles.		- Palpitation - lightheadedness - Weakness - Shortness of breath - Chest pressure

-
-

NORMAL HEART RATE/MINUTE FOR AGE

Age	Heart rate
Newborn	100–160
< 1 year	110–160
1–2 years	100–150
2–5 years	95–140
5–12 years	80–120
> 12 years	60–100

- **Signs and Symptoms**

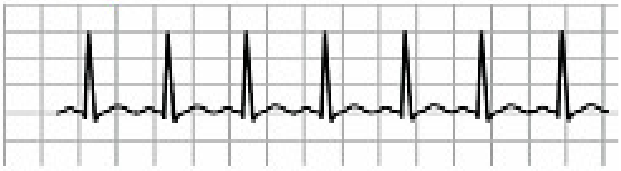
	Symptoms	*Signs*
Infants	Color changes (pale, mottled)	Irregular pulse
	Irritability	Tachycardia
	Feeding difficulties	Bradycardia
	Sweating	Signs of cardiac fail
	Tachypnoea/apnoeic	spells
Children	Chest Pain	Signs Of Cardiac F
	Dizziness	Tachycardia
	Palpitations	Bradycardia
	Fatigue	Syncope

- ***Note:*** *All patients with arrhythmias should be referred to a cardiologist*
-
-
- Investigations

- - ECG is essential for diagnosis, preferably a 12 lead ECG
- - Echocardiogram
- - Other according to the suspected etiology
-

TACHYARRHYTHMIAS

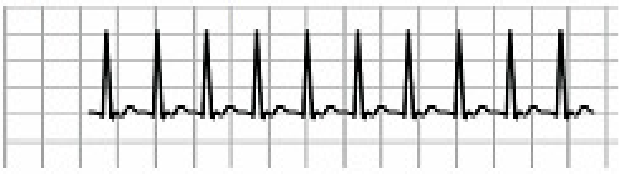
Sinus tachycardia



ECG Criteria

Rate: > upper limit for age
Rhythm: regular
P wave: present and normal
QRS: normal

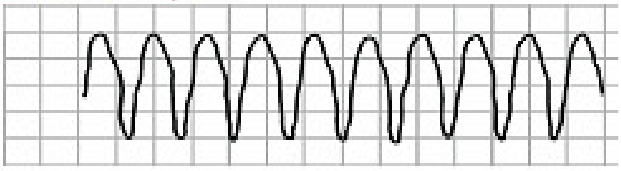
Supraventricular Tachycardia



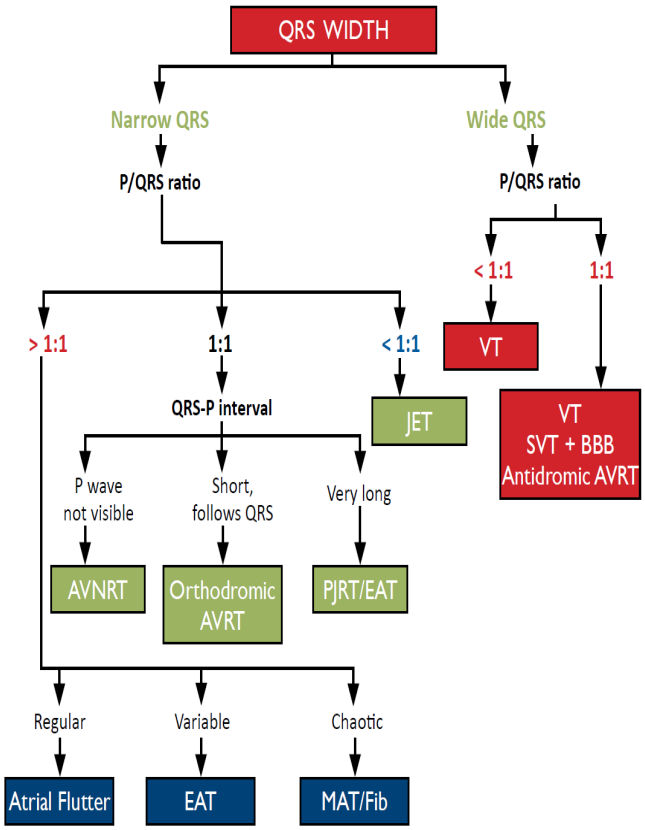
ECG Criteria

Rate: usually > 200 beats per minute
Rhythm: regular
P wave: abnormal
QRS: narrowed

Ventricular Tachycardia

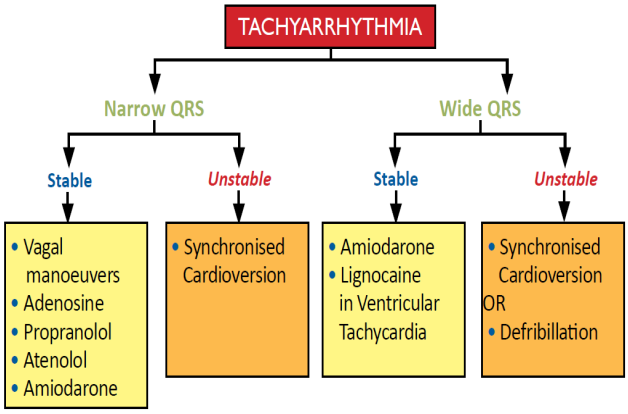


ALGORITHM FOR IDENTIFYING TACHYARRHYTHMIA



Abbrevations. VT, ventricular tachycardia; JET, junctional ectopic tachycardia; SVT, supraventricular tachycardia; BBB, bundle branch block; Fib, fibrillation. AVRT, atrioventricular re-entry tachycardia; AVNRT, atrioventricular nodal re-entry tachycardia; PJRT, permanent junctional reciprocating tachycardia; EAT, ectopic atrial tachycardia; MAT, multifocal atrical tachycardia;

ALGORITHM FOR MANAGEMENT OF ACUTE TACHYARRHYTHMIA



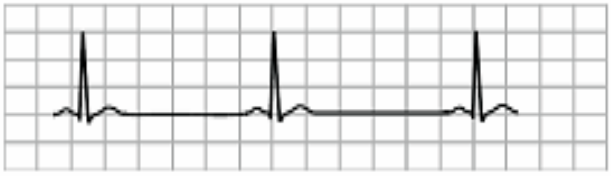
Pitfalls in management

- Consult a cardiologist if these acute measures fail to revert the tachycardia.
- In Wolff-Parkinson-White syndrome, digoxin is contraindicated because paroxysms of atrial flutter or fibrillation can be conducted directly into the ventricle.
- Adenosine unmasks the atrial flutter by causing AV block and revealing more atrial beats per QRS complex.
- In wide QRS complex tachycardia with 1:1 ventriculoatrial conduction, it is reasonable to see if adenosine will cause cardioversion, thereby making a diagnosis of a conduction system dependent SVT.
- A follow up plan should be made in consultation with cardiologist.

3.10. Bradyarrhythmias

ECG criteria	
Age Group	Heart Rate
Infants to < 3 years	<100 bpm
Children 3 – 9 years	< 60 bpm
Children 9 – 16 years	< 50 bpm
Adolescents > 16 years	< 40 bpm
24 hours Ambulatory ECG criteria	
Age Group	Heart Rate
Infants to 1 year of age	< 60 bpm sleeping, < 80 bpm av
Children 1 – 6 years	< 60 bpm
Children 7 – 11 years	< 45 bpm
Adolescents, young adults	< 40 bpm
Highly trained athletes	< 30 bpm

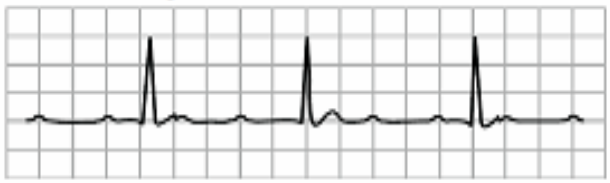
Sinus Bradycardia



ECG Criteria

Rate: < lower limit for age
Rhythm: regular
P wave: present, all look the same
QRS: normal, 80-120 millisecond

Heart Block (Complete)



- Causes
- - Hypoxia
- - Hypothermia
- - Head injuries and increased intracranial pressure
- - Toxins and drug overdose
- - Post operative
- - Congenital excessive vagal stimulation
- - Electrolyte disturbances (Hypo- or hyperkalaemia, Hypocalcaemia)
- Sinus Bradycardia
- ECG Criteria
- Rate: low, usually < 60 beats per minute P wave: independent P waves QRS's with no relationship between the two (AV dissociation)
- Management
- - If syncope and Heart rate - below 50/min
- • Start i.v. Isuprel (Isoprenaline) 0. 05 – 0. 4 microgram/kg/min.
- Or
- • Dobutamine (Dobutrex) 2 - 20 microgram/kg/min
- • Insert pacemaker if ineffective

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- **4. Haematological Conditions**

- 4.1. Anemia

- Definition: Anemia is defined as a reduction of the red blood cell
- (RBC) volume or hemoglobin concentration below the range of normal
- values occurring in healthy persons.
- Cause
- - Anemia is classified according to physiologic process (decreased production, increased destruction or blood loss). In practice, classifying anemia according MCV is a useful approach to assess the common causes of anemia in children.
- Signs and Symptoms
- - Pale mucous membranes, palms and nail beds
- - dizziness, fainting
- - Headache
- - Shortness of breath on exertion (exercise intolerance)
- - Palpitations
- - Visual disturbances
- - Poor growth
- - Confusion, decreased mental activity
- - Rapid heartbeat or palpitations
- - dyspnoea, tachypnea
- - Signs of cardiac failure if severe anemia

Mean Hemoglobin and hematocrit values by age

	Hb (g/dl)		Ht (%)		Reticulocytes *(%)	MCV (FL)	WBC		Platelets (10^3/mm^3)
Age	Mean	Range	Mean	Range	Mean	Lowest	Mean	range	Range
Cord blood	16.8	13.7-20.1	55	45-65	5.0	110	18000	9000-30000	290
2 weeks	16.5	13.0-20.0	50	42-66	1.0		12000	5000-21000	252
3 months	12	9.5-14.5	36	31-41	1.0		12000	6000-18000	150-350
6 months-6 years	12	10.5-14.0	37	33-42	1.0	70-74	10000	6000-15000	150-350
7 years-12 years	13	11.0-16.0	38	34-40	1.0	76-80	8000	4500-13500	150-350
Adults									
Female	14	12.0-16.0	42	37-47	1.6	80	7500	5000-10000	150-350
Male	16	14.0-18.0	47	42-52		80			150-350

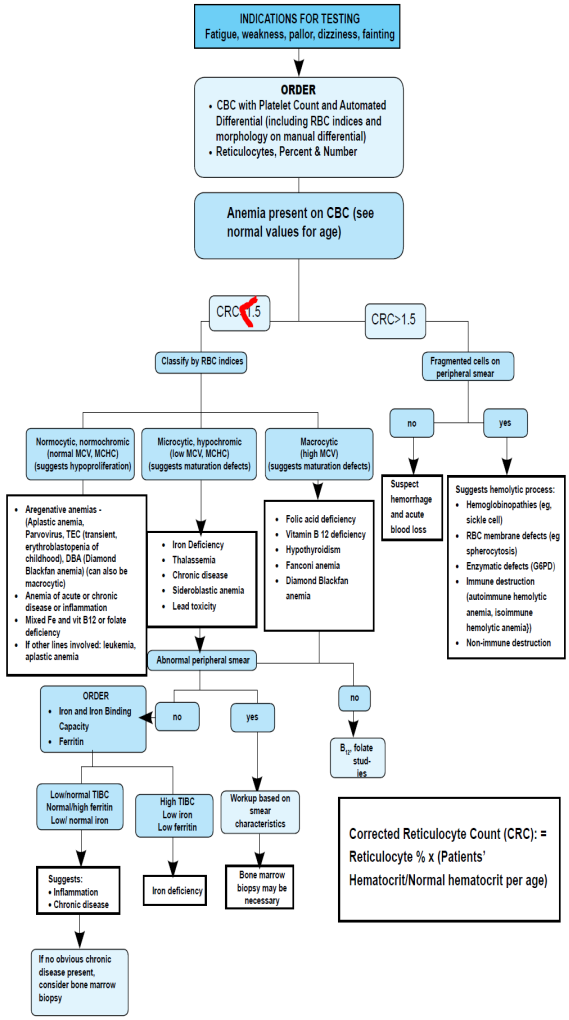
*= *normal reticulocyte count in a person who is not anemic. In a child who is anemic, always calculate the Reticulocyte index (or corrected reticulocyte count)*

-
- Normal values vary by age, sex and ethnicity. Means and ranges for hemoglobin and hematocrit values by age groups of well-nourished children
- Reticulocytes

are circulating immature RBC. Normal range are in the table above. However, if a person has anemia and his bone marrow is able to produce new blood cells, his/her reticulocyte percentage should be higher than

“normal” Thus, calculating the corrected reticulocyte count is an important step in understanding whether the reticulocyte count is appropriate or inappropriate to the situation.

- Corrected Reticulocyte Count (CRC): = Reticulocyte % x (Patients’ Hematocrit/Normal hematocrit per age)
- - A CRC >1.5 suggests increased Red Blood Cells production as a result of hemolysis and blood loss.



-
- Physiologic classification of Anemia
- - Anemia due to reduced red blood cell/hemoglobin production
- • Bone marrow aplasia:

- → Fanconi's anemia (congenital aplastic anemia)
- → Acquired aplastic anemia, diamond-Blackfan
- anemia, Transient Erythroblastopenia of childhood (red blood cell aplasia).
- • Bone marrow replacement by tumour cells - leukaemias,
- secondary metastases.
- • Bone marrow replacement by fibrous tissue or granulomas
- - granulomas can occur in the congenital toxoplasmosis, other, rubella, cytomegalovirus, herpes simplex (TORCH),
- infections (or in tuberculosis infection. Parvovirus is directly cytotoxic because it replicates in the erythroid precursors
- • deficiency of iron

Causes of Iron Deficiency Anemi
Chronic blood loss
Increase demand
• Prematurity
• Growth
Malabsorption
• Worm infestation
Poor diet

-
- • deficiency of folic acid: Megaloblastic anemia of infancy can develop due to:
- → Folic acid deficiency during rapid growth.
- → Malabsorption syndromes such as coeliac disease, in inflammatory bowel disease and in children taking anticonvulsants.
- • deficiency of vitamin B12 can occur in:
- → Infants who are breast-fed by a vegetarian mother
- → Mal-absorption
- → Worm infestation
- → Congenital pernicious anemia where there is inability to secrete gastric intrinsic factor
- • ***Thalassaemias:***

Introduction

• β-Thalassaemia major is an inherited blood disorder presenting with anaemia at 4 - 6 months of age.

• Common presenting symptoms are pallor, lethargy, failure to thrive and hepatosplenomegaly.
• In Malaysia, the β-thalassaemia carrier rate is estimated at 3-5%, most of whom are unaware of their carrier / thalassaemia minor status.
• The carrier rates of α-thalassaemia and Haemoglobin E (HbE) are 1.8-7.5% and 5-46% respectively. HbE are found more in the northern peninsular states.
• Interaction between a β-thalassaemia carrier with a HbE carrier may result in the birth of a patient with HbE/β-thalassaemia or thalassaemia intermedia with variable clinical severity.
• The moderate to severe forms behave like β-thalassaemia major patients while the milder forms are asymptomatic.
***Baseline investigations** to be done for all new patients: -*
• Full blood count, Peripheral blood film (In typical cases, the Hb is about 7g/dl)
• Haemoglobin analysis by electrophoresis / HPLC:
• Typical findings for β-thalassaemia major: HbA decreased or absent, HbF increased, HbA2 variable.
• Serum ferritin.
• Red cell phenotyping (ideal) before first transfusion.
• DNA analysis (ideal)
• For the detection of α-carrier and confirmation of difficult cases.

• Mandatory in prenatal diagnosis.
• Liver function test.
• Infection screen: HIV, Hepatitis B & C, VDRL screen (before first transfusion).
• HLA typing (for all patient with unaffected siblings)
• All nuclear family members must be investigated by Hb Analysis for genetic counselling.

- *• 1st degree and 2nd degree relatives should also be encouraged to be screened & counselled (cascade screening).*

Management

Regular maintenance blood transfusion and iron chelation therapy is the mainstay of treatment in patients with transfusion dependent thalassaemia.

. ***Maintenance Blood Transfusion***

Beta thalassaemia major
• When to start blood transfusion?
• After completing blood investigations for confirmation of diagnosis.
• Hb < 7g/dl on 2 occasions > 2 weeks apart (in absence other factors e.g. infection).
• Hb > 7g/dl in β+-thalassaemia major/severe forms of HbE-β-thalassaemia if impaired growth, para-spinal masses, severe bone changes, enlarging liver and spleen.
• Transfusion targets?

• Maintain pre transfusion Hb level at 9 -10 g/dl.
• Keep mean post-transfusion Hb at 13.5-15.5g/dl.
• Keep mean Hb 12 - 12.5 g/dl.
• The above targets allow for normal physical activity and growth, abolishes chronic hypoxaemia, reduce compensatory marrow hyperplasia
which causes irreversible facial bone changes and para-spinal masses.
• Transfusion interval?
• Usually 4 weekly interval (usual rate of Hb decline is at 1g/dl/week).

• Interval varies from individual patients (range: 2 - 6 weekly).
• Transfusion volume?
• Volume: 15 - 20mls/kg (maximum) packed red cells (PRBC).
• Round-up to the nearest pint of cross-matched blood provided. i.e. if calculated volume is just > 1 pint of blood, give 1 pint, or if calculated volume is just < 2 pints, give 2 pints.
This strategy minimizes the number of exposure to immunologically different units of blood product and avoid wastage of donated blood.
Note:
• In the presence of cardiac failure or Hb < 5g/dl, use lower volume PRBC (< 5ml/kg) at slow infusion rate over > 4 hours with IV Frusemide 1 mg/kg
(20 mg maximum dose).
• It is recommended for patients to use leucodepleted (pre-storage, post storage or bedside leucocyte filters) PRBC < 2 weeks old.
• Leucodepletion would minimize non-haemolytic febrile reactions and

alloimmunization by removing white cells contaminating PRBC.

Thalassaemia intermedia

• A clinical diagnosis where patients present later with less severe anaemia at > 2 years of age usually with Hb 8g/dl or more.

• Severity varies from being symptomatic at presentation to being asymptomatic until later adult life.

• Assessment and decision to start regular transfusion is best left to the specialist.

Alpha Thalassaemia (Hb H disease)

- Transfuse only if Hb persistently < 7g/dl and/or symptomatic.

Iron Chelation Therapy

- This is essential to prevent iron overload in transfusion dependent thalassaemia.
- Compliance to optimal treatment is directly related to superior sur outcome, now possible beyond the 6th decade.
- Currently 3 approved iron chelators are available: Desferrioxamine Deferiprone (DFP) and Deferasirox (DFX).

Desferrioxamine (Desferal®)

- When to start?
  - Usually when the child is > 2 - 3 years old.
  - When serum ferritin reaches 1000 µg/L.
  - Usually after 10 – 20 blood transfusions.
- Dosage and route
  - Average daily dose is 20 – 40mg/kg/day.
  - by subcutaneous (s.c.) continuous infusion using a portable pum 8-10 hours daily, 5 - 7 nights a week.
- Aim to maintain serum ferritin level below 1000 µg/L.
- Vitamin C augments iron excretion with Desferal®.
- Severely iron loaded patients require longer or continuous SC or IV (via Portacath) of Desferal®.

Complications of Desferal®

- Local skin reactions usually due to inadequately diluted Desferal® or in
- Yersinia infection: presents with fever, abdominal pain & diarrhoea

Oral iron chelator

- **Deferiprone / L1 (Ferriprox®/Kelfer®)** is an alternative if iron chelation is ineffective or inadequate despite optimal Desferal® use, or if Desferal® us is contraindicated. However, there is no formal evaluation in children < 10 years of age.
- Deferiprone is given 75 – 100 mg/kg/day in 3 divided doses.
- It can also be used in combination with Desferal®, using a lower dose of 50mg/kg/day.
- There are risks of GI disturbance, arthritis and rare occurrence of idiopath agranulocytosis.
- Weekly full blood count monitoring is recommended. Stop if neutropenic (<1,500/mm³).
- **Deferasirox (Exjade®)** can also be used for transfusional iron overload in patients 2 years or older but is expensive.
- The dose is 20-30 mg/kg/day in liquid dispersible tablet, taken once daily.
- There are risks of transient skin rash, GI disturbance and a reversible rise i serum creatinine.
- Monthly monitoring of renal function is required.

Monitoring of patients

During each admission for blood transfusion,
the following should be done:

- Clinical assessment: height, weight, liver & spleen size, any adverse side effects of chelation therapy.
- Pre-transfusion Hb, platelet count and WBC (if on Deferiprone).
- Post transfusion Hb – ½ hour post transfusion.
- Calculate the volume of pure RBC transfused based on the haematocrit (HCT) of packed red blood cells (PRBC) given (usually HCT of PRBC from blood bank is > 50 - 55%).
  - Volume of pure RBC transfused = volume of blood given x HCT of PRBC given (e.g. 600 mls x 0.55 = 330 mls).

Every year or more frequent if indicated

- Evaluate growth and development
- Endocrine assessment – modified GTT, T4/TSH, Ca, PO4
  (If Ca low - check PTH & Vit. D).
- Pubertal and sexual development from 10 years onwards.
- Tanner stage of breast and genitalia.
- Follicle stimulating hormone (FSH), luteinizing hormone (LH) levels,
  oestradiol or testosterone hormone levels.
- Infection screen (6 monthly) – Hepatitis B and C, HIV, VDRL.
- Annual volume of pure red blood cell transfused/median body weight.
- Evaluate iron balance and overload status.
- Bone: osteoporosis & skeletal abnormalities.

Cardiac assessment at variable intervals and especially after 10 years of age

- Yearly ECG or Holter monitoring for arrhythmias.
- Annual cardiac echocardiography.
- Cardiac T2* MRI.

Liver iron assessment

- Liver T2* MRI for non-invasive assessment of liver iron.
- Liver biopsy for liver iron concentration and the assessment of hepatitis,
  fibrosis or cirrhosis in selected cases and prior to bone marrow
  transplantation.

Splenectomy

Indications

- Blood consumption volume of pure RBC > 1.5X normal or
  >200-220 mls/kg/year in those > 5 years of age to maintain average

Diet and supplements

- Oral folate at minimum 1 mg daily may benefit most patients.
- Low dose Vitamin C at 3 mg/kg augments iron excretion for those on Desferral only.
  - Dose: <10 yrs, 50mg daily; >10yrs, 100mg daily given only on deferral days
- Avoid iron rich food such as red meat and iron fortified cereals or milk.
- Tea may help decrease intestinal iron absorption.
- Dairy products are recommended as they are rich in calcium.
- Vitamin E as antioxidant.
- Calcium and zinc.

Bone marrow transplantation (BMT)

- Potential curative option when there is an HLA-compatible sibling donor.
- Results from matched unrelated donor or unrelated cord blood transplant are still inferior with higher morbidity, mortality and rejection rates.
- Classification of patients into Pesaro risk groups based on the presence of 3 risk factors: hepatomegaly > 2cm, irregular iron chelation and presence of liver fibrosis.
- Best results if performed at the earliest age possible in Class 1 patients.

Pesaro Risk Groups and Outcome following BMT

Class	No. of risk factors	Event Free Survival %	Mortality %	Rejection %
I	0	91	7	2
2	I-2	83	13	3
3	3	58	21	28
Adults	-	62	34	-

Note: In newly diagnosed transfusion dependent thalassaemics, the family should be informed of this option and referred early to a Paediatrician for counselling and HLA typing of patient and unaffected siblings to identify a potential donor.

Antenatal diagnosis

- - Anemia of chronic disease (e.g: chronic pyelonephritis, chronic renal failure, bacterial endocarditis, osteomyelitis) due to:
- • Impaired erythropoietin production.
- • Anemia can also be associated with hypothyroidism.
- • Sideroblastic anemia (Extremely rare, heterogeneous group of diseaeses, either acquired (drugs, toxins, malignancy), or congenital . diagnosis by bone marrow
- biopsy (presence of sideroblasts) anemia
- - Anemia due to increased red blood cell destruction (haemolysis)
- • Congenital
- → Red cell membrane defects - including hereditary spherocytosis.
- → Red cell enzyme abnormalities - including glucose-6-phosphate dehydrogenase (G-6-Pd) deficiency, pyruvate kinase deficiency.

- → Hemoglobinopathies - including sickle cell disease, thalassaemias.
- • Acquired
- → Autoimmune haemolysis
- → Isoimmune haemolysis (haemolytic disease of the newborn, blood transfusion reactions).
- → Infections (including malaria, septicaemia)
- → drug- and toxin-induced
- → disseminated intravascular coagulation
- → Hypersplenism.
- - Anemia due to blood loss
- • Including gastrointestinal blood loss, traumatic, heavy menstruation in girls.
- Complications
- - Pulmonary edema
- - Congestive heart failure
- - Acute respiratory distress syndrome (ARdS)
- Investigations according to clinical situation
- - FBC and reticulocyte count and peripheral blood smear

- examination
- - Blood film for malaria parasites
- - Stool examination for eggs of hookworm / Stool for occult blood,
- ova and parasites
- - Sickling test
- - Hemoglobin electrophoresis
- - Analysis for nutritional deficiencies
- - Bone marrow aspiration to assess the decreased production of red cells
- - Coombs direct and indirect (in cases of hemolytic anemia)
- - Iron studies (Fe, Ferritin, TIBC, transferring % saturation)
- - Other investigations will be dependent on the clinical evaluation of the patient
- Management
- - Obtain a detailed history from the patient or care givers
- - Examine the anaemic patient carefully and perform the
- appropriate investigations with a goal of:
- • Confirming that the patient is anaemic

- • Establishing the type of anemia
- • determining the cause of the anemia
- • determining whether or not there are complications arising from the anemia, the cause of the anemia or both
- • Remove or correct the underlying cause
- • Always investigate cause of anemia before initiating treatment
- • In an emergency, take all blood samples before treatment
- Therapeutic objectives
- • Treat underlying cause of anemia
- • In sickle cell disease patients restore hemoglobin to steady state level
- • In iron deficiency replenish iron stores after correction of anemia (continue to treat for 2-3 months)
- Non-Pharmaceutical
- • Advise on a balanced diet especially iron-rich foods such as liver; beef kidneys; molasses; meat; sardines; eggs, fish; fresh green leafy vegetables..

- • Malaria prevention
- • Encourage exclusive breastfeeding until 6 months, then supplementation with iron rich food. discourage use of cow's milk before 12 months and excessive intake of cows milk.
- Pharmaceutical management
- • For iron deficiency anemia, prescribe Elemental Iron
- 4-6 mg/kg/day divided in 3 doses daily until the Hb has reached the normal range. Pay attention to type of iron supplementation prescribed (Ferrous Sulphate has 20% elemental iron, Ferrous Fumarate has 33% elemental iron
- and Ferrous gluconate has 12% elemental iron). Continue
- for 2-3 months after normalization of Hb to build up iron stores.
- • Sickle cell disease patients should receive iron tablets only if there is evidence of iron deficiency. They should however, receive Folic acid. Similarly, patients whose anemia is possibly

due to malaria should receive folic acid

- • Folic acid, oral: 5 mg every 2 days for 30 days or for as long as required.
- • If anemia is due to hookworms treat appropriately (Albendazole 400 mg po x 3 days or mebendazole 100 mg po x 3 days)
- • Vitamin B12 deficiency: (Hydroxycobalamin) injection IM:
- Initially 100mcg/day X 10-15 days. Maintenance dose 30-50 mcg/month. Lifelong treatment may be required.
- • Severe anemia with signs of cardiac failure will need treatment of the heart failure in addition to bloodTransfusion with packed cells. Look for signs of decompensation before deciding to transfuse and look for
- these signs during transfusion.
- Transfuse the patient if Hb < 5 g/dl and decompensation
- signs are present: Packed cells: 10-15 ml/kg body weight

- slowly over 4 hours and Whole blood: 20ml/kg body weight
- Side effects of iron therapy
- • diarrhea, abdominal discomfort, constipation, or black Stools
- Recommendations
- - Refer all patients with anemia related to poor diet to a nutritionist or a health center for nutritional follow-up
- - Refer all patients with recurrent anemia or with anemia of unknown cause to a referral hospital
    - **4.2. Sickle Cell Anemia**
- Definition: Chronic haemolytic anemia characterized by sickle–shaped Red blood cells as a result of mutation in the β chainHemoglobin
- Cause
- - Homozygous inheritance of mutated HBS (amino–acid valine is substituted for glutamic acid in the position 6 of the β–chain)
- Signs and Symptoms
- - Impaired growth and development
- - Anemia and mild jaundice

- - Hepatosplenomegaly (in younger children)
- - Bone pain (especially long bones in children)
- - Pain and swelling of the hands and feet (hand - foot syndrome)
- in children between 6 months and 3 years old.
- - Arthralgia with fever
- - Severe abdominal pain with vomiting
- - Acute Chest Syndromes (sudden onset of fever, cough, chest
- pain, tachypnea leucocytosis and pulmonary infiltrates on x–
- ray): Must be aggressively treated may be fatal
- - Tower shaped ("frontal and parietal bossing") skull
- Complications
- - Infections (especially from encapsulated organism such as
- Streptococcus pneumoaniea:
- • Osteomyelitis (Streptotococcus pneumonia and Salmonella)
- • Meningitis
- - Aplastic crisis (Infection by Parvovirus B19 that infects RBC

- progenitors resulting in a very rapid drop in Hb).
- - Stroke (infarctive) with hemiparesis and convulsions
- - Gangrene (vaso-occlusive)
- - Pulmonary hypertension
- - Acute chest syndrome (sudden onset of fever, cough, chest pain,
- tachypnea leucocytosis and pulmonary infiltrates on x−ray):
- Must be aggressively treated as may be fatal
- - Gall bladder stones +/- cholecystitis
- - Splenic Sequestration (in 5 first years of life): onset of life
- threatening anemia with rapidly enlarging spleen and high reticulocyte counts
- - Avascular necrosis of the femoral head is common
- - Occlusion of major intracranial vessels may lead to hemiplegia
- - Cranial nerve palsies and other neurological deficits
- - Priapism
- Investigations
- - Full blood count

- - Peripheral blood thick smear
- - Sickling test (Test d'Emmel)
- - Hb electrophoresis
- - X-ray of long bones, cortical thinning
- - X-ray of skull bone (shows widening of diploic space)
- Management
- Aims at three types of crisis
- • Thrombotic (vaso–occlusive, painful or infarctive)
- • Aplastic (sequestration)
- • Haemolytic
- Non-pharmacological
- • IV or oral fluids 2L/m2/day
- • Oxygen if in respiratory distress
- Pharmaceutical
- For complications
- • Analgesics (WHO Step wise pain management)
- → Paracetamol 10-15mg/kg/dose po every 4-6 hours
- associated with Brufen 5-10mg/kg/dose po every 6-8 hours
- → codeine 0.5-1mg/kg/dose every 6 hours
- → Pethidine 0.5–2mg/kg 4hrly

- → Morphine (titrate to effect) PO: 0.2-0.5 mg/kg/dose
- every 4-6 hours, IV, IM, SC: 0.1-0.2 mg/kg/dose every 2-4 hours
- • If patient has an infection treat according to the bacteria, the site and the severity of the infection
- • Aggressively search for cause of infection (hemoculture, urine culture, chest X ray) and start empiric antibiotic treatment if child sick with fever
- • Blood Transfusion: A child with sickle cell disease has chronic anemia which is usually well tolerated.
- → Transfusion should be reserved for the following
- circumstances:
- □ Urgently for sudden, severe anemia due to acute splenic sequestration, Parvovirus B19 infection, or hyper hemolytic crises.
- □ In acute chest syndrome and perioperatively.

- → Acute red cell exchange transfusion is indicated in the following situations
- □ Acute infarctive stroke
- □ Severe acute chest syndrome
- □ Multiorgan failure syndromes
- □ Priapism that does not resolve after adequate
- hydration and analgesia
- • Additional treatment
- → Give supplementary Folic Acid (5 mg oral daily) but avoid iron (risk of hemochromatosis).
- → Hydroxyurea should be given to patients with more than 3 crises per year. Start at a dose of 10 mg/kg PO daily and titrate by 5mg/kg every 8 to 12 weeks to a maximum dose of 25mg/kg/day.
- → Homozygous should be vaccinated for salmonella, Pneumococcal and Haemophilus influenzae
- Recommendations
- - Education of patient on sickle cell disease and crisis to avoid complications
- • Should drink much water daily

- • Avoid getting cold (dress with warm clothes by cold weather)
- - Sickle cell screening before marriage for suspected carriers and genetic counseling if possible
- - Heterozygote carriers should have family members screened for sickle cell disease

- 4.3. Idiopathic Thrombocytopenic Purpura (ITP)

- Definition: Idiopathic Thrombocytopenic Purpura (also called immune thrombocytopenic purpura), is a blood – clotting disorder that can lead to easy or excessive bruising and bleeding. Children often develop ITP after a viral infection and usually recover fully without treatment.

Pathogenesis

• Increased platelet destruction, likely due to autoantibodies to platelet membrane antigens.

- • In children, ITP is an acute, self-limiting disorder that resolves spontaneously.

Clinical Manifestations

• Onset is usually acute.
• Majority will give a history of a viral infection in the preceding 2-4 weeks
• Spectrum of bleeding severity ranges from cutaneous bleeding
    - i.e. petechiae, to mucosal bleeds i.e. gum bleeds and epistaxis, to life threatening bleeds i.e. intracranial haemorrhage.

Diagnosis and Investigations

• Diagnosis is based on history, physical examination, blood counts, and examination of the peripheral blood smear.
• Physical examination: absence of hepatosplenomegaly or lymphadenopathy.
• Blood counts: isolated thrombocytopenia, with normal haemoglobin and white cell count.
• Peripheral blood picture: normal apart from reduced, larger platelets, no abnormal cells.
• Threshold for performing a bone marrow aspiration is low and is indicated:
• Before starting steroid therapy (to avoid partially inducing an undiagnosed acute leukaemia).

• If there is failure to respond to Immunoglobulin therapy.
• When there is persistent thrombocytopenia > 6 months.
• Thrombocytopenia recurs after initial response to treatment.

- • Other tests that may be indicated when there is atypical presentation are:

Antinuclear factor and DNA antibodies.
• Coomb's test.
• CMV serology for those less than a year old.
• Coagulation profile for those suspected non-accidental injury and inherited bleeding disorder.
• HIV testing for those at risk i.e. parents who are HIV positive or intravenous drug users.

- • Immunoglobulin levels for those with recurrent infections.

Other causes of Thrombocytopenia
Neonatal alloimmune/ isoimmune
• Thrombocytopenia if < 6 months old
Sepsis and infections including HIV infection
Drug-induced thrombocytopenia
Haematological malignancy
• e.g. Acute leukaemias
Congenital marrow failure syndromes
• e.g. Fanconi anaemia, thrombocytopenia with abser
Autoimmune disorders
• e.g. Systemic lupus erythematosus, Evan syndrome
Primary immunodeficiency syndromes
• e.g. Wiskott-Aldrich syndrome

-
- Differential diagnosis: (ITP is a diagnosis of exclusion)
- - Systemic Lupus Erythematosus (SLE),
- - HIV infection

- - Wiskott-Aldrich Syndrome (WAS)) must be considered in young males found to have low platelet counts, particularly if there is a history of eczema and recurrent infection.

Management

• Not all children with diagnosis of acute ITP need hospitalization.

• Hospitalization is indicated if:

• There is severe life-threatening bleeding (e.g. ICH) regardless of platelet count.

• Platelet count < 20 x 109/L with evidence of bleeding.

• Platelet count < 20 x 109/L without bleeding but inaccessible to health care.

• Parents request due to lack of confidence in homecare.

• Most children remit spontaneously: 70% achieve a platelet count > 50 x 10^9/L by the end of the 3rd week. Treatment should be individualised.

• Precautions with physical activities, avoidance of contact sports and seeking immediate medical attention if bleeding occurs should be advised.

• Careful observation and monitoring of platelet count, without specific treatment, is appropriate for patients with:
• Platelet count > 20 x 109/L without bleeding.
• Platelet count > 30 x 109/L with only cutaneous purpura.
• A repeat blood count should be performed within the first 7-10 days to ensure that there is no evidence of serious evolving marrow condition.
• Treatment is indicated if there is:
• Life threatening bleeding episode (e.g. ICH) regardless of platelet count.
• Platelet count < 20 x 109/L with mucosal bleeding.

- • Platelet count < 10 x 109/L with any bleeding.

• Choice of treatment includes:
• Oral Prednisolone 2 mg/kg/day for 14 days then taper off.
• Oral Prednisolone 4 mg/kg/day for 4 days.
• IV Immunoglobulin (IVIG) 0.8 g/kg/dose for a single dose.

Notes regarding treatment:

• All above are effective in raising platelet count much quicker compared to no treatment. However there is no evidence that these treatment
regimens reduce bleeding complications or mortality or influence progression to chronic ITP.
• Side effects of IVIG are common (15 - 75%): fever, flushing, headache, nausea, aseptic meningitis and transmission of Hepatitis C (older
preparations).
• Steroids should not be continued if there is no response or if there is a rapid relapse after withdrawal. The long-term side-effects in a growing
child outweigh the benefits of either frequent high-dose pulses or titration of platelet count against a regular lower steroid dose.
• Treatment should not be directed at increasing the platelet count above a preset level but rather on the clinical status of the patient (treat the child
and not the platelet count).

Intracranial Haemorrhage (ICH)

• The most feared complication of ITP.
• Incidence of ICH in a child with ITP is very low between 0.1 - 0.5%.
• The risk of ICH highest with platelet count < 20 x 109/L, history of head trauma, aspirin use and presence of cerebral arteriovenous malformation.
• 50% of all ICH occurs after 1 month of presentation, 30% after 6 months.
• Early treatment with steroid or IVIG may not prevent late onset ICH.

Emergency treatment

Emergency treatment of ITP with severe bleeding i.e. severe epistaxis or GIT bleed causing drop in Hb or ICH (alone or in combination) includes:
• High dose IV Methylprednisolone 30 mg/kg/day for 3 days.
• IVIG 0.8g - 1g/kg as a single dose.
• Combination of IVIG and methylprednisolone in life threatening conditions.
• Platelet transfusion in life threatening haemorrhage: 8 - 12 units/m2 body surface area (2 to 3 folds larger than usual units) as the platelets will be

consumed by the haemorrhage to form blood clots and will reduce further circulating platelets.

• Consider emergency splenectomy if other modalities fail.

- • Neurosurgical intervention in ICH, if indicated and to perform with splenectomy if necessary.
- Note MANAGEMENT: (for newborns, see national neonatology protocols)

CHRONIC ITP

Definition

• Persistent thrombocytopenia after 6 months of onset (occurs in 20%)

• Wide spectrum of manifestations: mild asymptomatic low platelet counts to intermittent relapsing symptomatic thrombocytopenia to the rare stubborn and persistent symptomatic and haemorrhagic disease.

Management

Counselling and education of patient and caretakers regarding natural history of disease and how to detect problems and possible complications early are important. Parents should be comfortable of taking

care of patients with persistent low platelet counts at home. At the same time they must be made aware of when and how to seek early medical attention when the need arises.

- Every opportunity should be given for disease to remit spontaneously as the majority will do so if given enough time.
- Revisit diagnosis to exclude other causes of thrombocytopenia (Immunodeficiency, lymphoproliferative, collagen disorders, HIV infection).
- Asymptomatic children can be left without therapy and kept under observation with continued precautions during physical activity.
- Symptomatic children may need short course of treatments as for acute ITP to tide them over the “relapse” period or during surgical procedures.

For those with Persistent bleeding, Second line therapies includes:
• Pulses of steroids: oral Dexamethasone 1 mg/kg given on 4 consecutive days every 4 weeks for 4 months.
• Intermittent anti-Rh(D) Immunoglobulin treatment for those who are Rhesus D positive: 45 - 50 ug/kg. May cause drop in Hb levels.
• ***Second line therapy should only be started after discussion with a Paediatric haematologist.***
Note:
• Care must be taken with any pulse steroid strategy to avoid treatment-related steroid side effects.
• Family and patient must be aware of immunosuppressive complications e.g. risk of severe varicella.
• There is no justification for long-term continuous steroids.
If first and second-line therapies fail, the patient should be managed by a paediatric haematologist.
Other useful agents are Rituximab and Cyclosporine.

Splenectomy

• Rarely indicated in children as spontaneous remissions continue to occur up to 15 years from diagnosis.
• The risk of dying from ITP is very low - 0.002% whilst the mortality associated with post-splenectomy sepsis is higher at 1.4 - 2.7 %.
• *Justified when there is:*
• Life-threatening bleeding event
• Severe life-style restriction with no or transient success with intermittent IVIG, pulsed steroids or anti-D immunoglobulin.
• *Laparoscopic method* preferred if expertise is available.
• Pre-splenectomy preparation of the child with immunization against pneumococcus, haemophilus and meningocccus must be done and post-splenectomy life-long penicillin prophylaxis must be ensured.
• Pneumococcal booster should be given every 5 years.
• Up to 70% of patients achieve complete remission post-splenectomy.

Haemophilia

Definition

- A group of blood disorders in which there is a defect in the clotting mechanism.
- Of X-linked recessive inheritance, but in 30% there is no family history as it is a spontaneous new mutation.
- The most common haemophilias are:
  - Haemophilia A – Deficiency of factor VIII (85% cases)
  - Haemophilia B – Deficiency of factor IX (15% cases)

Clinical Manifestation

- Bleeding in the neonatal period is unusual.
- Usually presents with easy bruising when crawling and walking (9-12 months age).
- Haemarthrosis is characteristic of haemophilia. Large joints are usually affected (knee, ankle, elbow); swollen, painful joints are common.
- Epistaxis, gum bleeding, haematuria also occur.
- Intracranial haemorrhages can be life threatening.
- Bleeding may also occur spontaneously or after trauma, operation or dental procedures.

Diagnostic Investigations

- Full blood count
- Coagulation screen: PT, APTT
- Specific factor assay: FVIII level (low in Haemophilia A).
- Specific factor assay: FIX level (low in Haemophilia B).
- Bleeding time if applicable.
- Von Willebrand screen even if APTT normal.

In haemophilia, the activated partial thromboplastin time (APTT) is prolonged in moderate and severe haemophilia but may not show prolongation in mild haemophilia. The platelet count and prothrombin time (PT) are normal. When the APTT is prolonged, then the lab will proceed to do the factor VIII antigen level. If this is normal, only then will they proceed to assay the Factor IX level. Once the level has been measured, then the haemophilia can be classified as below.

Classification of haemophilia and clinical presentation		
Factor level	**Classification**	**Clinical presentation**
< 1 %	Severe	Spontaneous bleeding, risk of intracranial haemorrhage
1-5 %	Moderate	Bleeding may only occur with trauma, surgery or dental procedures
5-25 %	Mild	

Further Investigations

- Hepatitis B surface antigen, anti HBS antibody
- Hepatitis C antibody
- HIV serology
- Renal profile and Liver function test.
- Platelet aggregation if high suspicion of platelet defect.
- Diagnosis of carrier status for genetic counseling.
  - Mother of a newly diagnosed son with haemophilia.
  - Female siblings of boys with haemophilia.
  - Daughter of a man with haemophilia.

Once a child is diagnosed to have haemophilia, check the viral status at diagnosis and then yearly. This is because treatment carries the risk of acquiring viruses. All haemophiliacs should be immunized against Hepatitis B.

Treatment

- Ideally, treatment of severe haemophilia should be prophylactic to prevent arthropathy and ensure the best quality of life possible.The dosage of prophylaxis is usually 25-35 U/kg of Factor VIII concentrate, given every other day or 3 times a week. For Factor IX, the dosage is 40-60 U/kg, given every 2-3 days. However, this form of management is costly and requires central venous access.
- On demand treatment is another treatment option when clotting factors are inadequate. It consists of replacing the missing factor: Factor VIII concentrates are used in haemophilia A, Factor IX concentrates in Haemophilia B. Fresh frozen plasma and cryoprecipitate ideally SHOULD NOT be used as there is a high risk of viral transmission.
- The dose of factor replacement depends on the type and severity of bleed.

Suggested Replacement Doses of Factor VIII and XI Concentrate		
Type of bleed	Factor VIII dose	Factor XI dose
Haemarthrosis	20 U/kg	40 U/kg
Soft tissue or muscle bleeds	30-40 U /kg	60-80 U/kg
Intracranial haemorrhage or surgery	50 U/kg	100 U/kg

- Dose of factor required can also be calculated using the formulas below
  - Units of Factor VIII: (% rise required) x (weight in kg) x 0.5.
  - Units of Factor IX: (% rise required) x (weight in kg) x 1.4.
- The percentage of factor aimed for depends on the type of bleed.
  - For haemarthroses, 30-40 % is adequate.
  - For soft tissue or muscle bleed aim for 40- 50 % level.
    (there is potential to track and cause compression/compartment syndrome)
  - For intracranial bleeds or patients going for surgery, aim for 100%.
- Infuse Factor VIII by slow IV push at a rate not exceeding 100 units per minute in young children.

Immunisations

- This is important and must be given: The subcutaneous route is preferred.
- Give under factor cover if haematomas are a problem.

Haemophilia Society

- All haemophiliacs should be registered with a patient support group e.g. Haemophilia Society.
- They should have a medic-alert bracelet/chain which identifies them as haemophiliacs and carry a book in which the diagnosis, classification of severity, types of bleeds and admissions can be recorded

SPECIFIC GUIDELINES FOR MANAGEMENT

Intracranial haemorrhage (ICH)

- Give factor replacement before suspected bleed is confirmed by CT scan
- Aim to increase Factor VIII level to 100%.
- For haemophilia B if monoclonal factor IX is used a level of 80% is adequate and if prothrombin complex concentrate (PCC) is used 50% level is recommended.
- Urgent CT scan:
  - If CT scan confirms ICH : maintain factor level 80%–100% for 1–7 days and 50% for 8–21 days.
  - If CT scan show no evidence of ICH, admit 1 day for observation.
- Follow up for long term sequelae.
- Lab investigations:
  - Pre-treatment factor assay level and inhibitor level before starting treatment and to repeat after 3 days of treatment to ensure adequate levels have been achieved and no inhibitor has developed.
  - Post treatment factor assay level (½ hour after infusion) to ensure required factor level is achieved (if the level is not achieved , consider development of inhibitors) and should be repeated after 3 – 5 days.
- follow up CT scan after 2 weeks

Surgery

- Pre-op investigations
  - Full coagulation profile – PT, PTT
  - Pre-factor assay level and inhibitor level
  - Blood grouping, full antibody screening and full cross matching if required.
- Calculate dose
  - ½ hour before operation, infuse patient with appropriate factors.
  - Preferable level :
    - 80-100% for factor VIII
    - 70% for monoclonal factor IX
    - 50% if prothrombin complex concentrate (PCC) used
- Check post transfusion specific factor level ½ hour later if necessary or after surgery to ensure correct factor level is achieved.

- Clotting factor level should be maintained above 50% during the operation and 24 hours after surgery.
- Maintain adequate factor levels -
  - Days 1-3 60-80%
    4-7 40-60%
    8-14 30-50%
- Repeat factor assay and check inhibitor level on day 3 to ensure adequate. levels. Post operatively a minimum of 10 to 14 days replacement therapy is recommended.

Iliopsoas bleed

- *Symptoms*: Pain/discomfort in the lower abdomen/upper thighs
- *Signs*: Hip flexed, internally-rotated, unable to extend
- *Danger*: Hypovolaemia, large volumes of blood may be lost in the retroperitoneal space.

Management:

- Factor replacement: 50U/kg stat, followed by 25U/kg bd till asymptomatic, then 20U /kg every other day for 10-14 days.
- Ultrasound / CTscan to diagnose.
- Physiotherapy - when pain subsides.
- Repeat U/S to assess progress.

Haematuria

Management

- Bed rest.
- Hydration (1.5 x maintenance).
- Monitor for first 24 hours: UFEME & Urine C&S.
- If bleeding persists for > 24 hours, start factor concentrate infusion.
- Perform KUB & Ultrasound of the kidneys.

DO NOT give anti-fibrinolytic drugs (tranexamic acid) because this may cause formation of clots in the tubules which may not recanalize.

Haemarthroses (Joint haemorrhages)

- Most spontaneous haemarthroses respond to a single infusion of factor concentrate. Aim for a level of 30 % to 40%.
- If swelling or spasm is present, treatment to level of 50% is required and infusion may have to be repeated at 12-24 hours interval until pain subsides.
- Minor haemarthroses may not require immobilization, elastic bandage or slings and ice may help in pain relief.
- Severe haemarthroses
  - Splint in position of comfort.
  - Rest.
  - Early physiotherapy.

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- 5. Endocrine System Conditions

- 5.1. Diabetes Mellitus (Type I and Type II)

- Definition: diabetes mellitus is a disorder of absolute or relative insulin deficiency that results in increased blood glucose and disruption of energy storage and metabolism. diabetes Mellitus is generally divided into two classifications: diabetes Mellitus I and diabetes Mellitus Type II.
- - diabetes Mellitus Type I: This results from the destruction of the pancreatic beta cells that leads to absolute insulin deficiency.
- Type IA is secondary to the autoimmune destruction of the beta cells. Type IB is secondary to non-autoimmune destruction of the beta cells. Type I diabetes accounts for approximately 2/3 of the new diagnosis of diabetes in patients < 19 years old. There is a component of genetic susceptibility and close relatives of patients with type I dM are at higher risk of developing the disease.
- - diabetes Mellitus Type II: This is secondary to varying degrees of insulin resistance and insulin deficiency and is related to both genetic and environmental influences including predisposing medication such as steroids and some ARVs. It is the most common type of diabetes mellitus in adults.
- - Neonatal diabetes: This is defined as persistent hyperglycemia occurring in the first months of life that lasts more than 2 weeks and requires insulin therapy for management. It is a rare cause of hyperglycemia in the neonate and has an estimated incidence of 1/500,000 births. The majority of affected infants are small for
- gestational age and present with weight loss, volume depletions, hyperglycemia and glucosuria with or without ketonuria and ketoacidosis.
- Signs and Symptoms
- - Polyuria: This occurs when the serum glucose concentration rises above 180 mg/dL exceeding the renal threshold for glucose and leads to increased urinary glucose excretion and a subsequent
- osmotic diuresis. This may present as nocturia, bedwetting, or daytime incontinence in a previously toilet trained child, or heavy diapers.
- - Polydipsia: This is secondary to increased thirst from increased serum osmolality and dehydration.
- - Polyphagia: This is due to an increased appetite that's initially secondary to loss of calories from glycosuria. These symptoms are not always present.

- - Weight loss: This is due to hypovolemia and increased catabolism.
- - Weakness/Lethargy with ultimate progression to coma: This is secondary to hypovolemia and electrolyte disturbances including progressive acidosis.
- Visual disturbances: This is secondary to osmotic changes in the lens.
- Complications
- - Short-term complications:
- • diabetic ketoacidosis (dKA): Occurs more frequently in type I diabetes mellitus, but may also occur in some forms of type I diabetes mellitus.
- • Hyperosmolar hyperglycaemic state (HHS): Occurs in type II diabetes mellitus.
- • Insulin resistance secondary to hyperglycemia: This occurs in both type I and type II diabetes mellitus.
- • Infections due to immunosuppression commonly include oral and vaginal candidiasis and Urinary Tract Infections.
- • death: Patients presenting with dKA or HHS have a high mortality rate.
- - Long Term complications:
- • Vascular complications including both micro-angiopathy and macro-angioapthy:
- →Nephropathy
- → Retinopathy
- → Neuropathy
- → Cardiovascular disease
- → Hypertension
- • dyslipidemia
- • Growth retardation or obesity depending on the insulin therapy. Patients may also have delayed puberty secondary to poor growth.
- • Psychiatric disorders including depression related to their chronic disease.
- Investigations
- - Blood sugar: The diagnosis is made based on abnormalities of the blood glucose. See diagnostic criteria below.
- - Additional studies to evaluate severity and complications of the disease:
- • Blood gas if concern for diabetic ketoacidosis.

- • Electrolytes
- • Renal function tests (urea and creatinine) to evaluate for diabetic nephropathy and dehydration.
- • Urine analysis to check for glycosuria, ketones, and protein
- • HbA1c: This can be used for diagnosis (see below) or to assess severity of disease and to assess response to therapy.
- • Lipid profile
- • Fundoscopy: This is to evaluate for diabetic retinopathy.
- • Foot examination: This is to evaluate for diabetic neuropathy and assess for wounds that may already be present.
- • Further history and physical examination to exclude other co-existing autoimmune diseases such as hypothyroidism,
- vitiligo, rheumatoid arthritis, etc., and to further ask about family history of endocrinopathies or autoimmune diseases.
- • Thyroid-stimulating hormone (TSH): This should be performed in type I diabetics as autoimmune diseases may occur together.
- Diagnosis criteria for diabetes mellitus

DIABETES MELLITUS (DM)
Symptoms of DM plus random plasma glucose ≥200 mg/dL (11.1 mmol/L) ***Or*** Fasting plasma glucose ≥126 mg/dL (7.0 mmol/L). Fasting is defined as no oral intake of foods for at least 8 hours. ***Or*** Two-hour plasma glucose ≥200 mg/dL during an oral glucose tolerance test (OGTT) as described by the WHO. ***Or*** HgA1C ≥ 6.5% This test should be performed in a certified laboratory with an assay standardized to the diabetes control and complications trial (DCCT).

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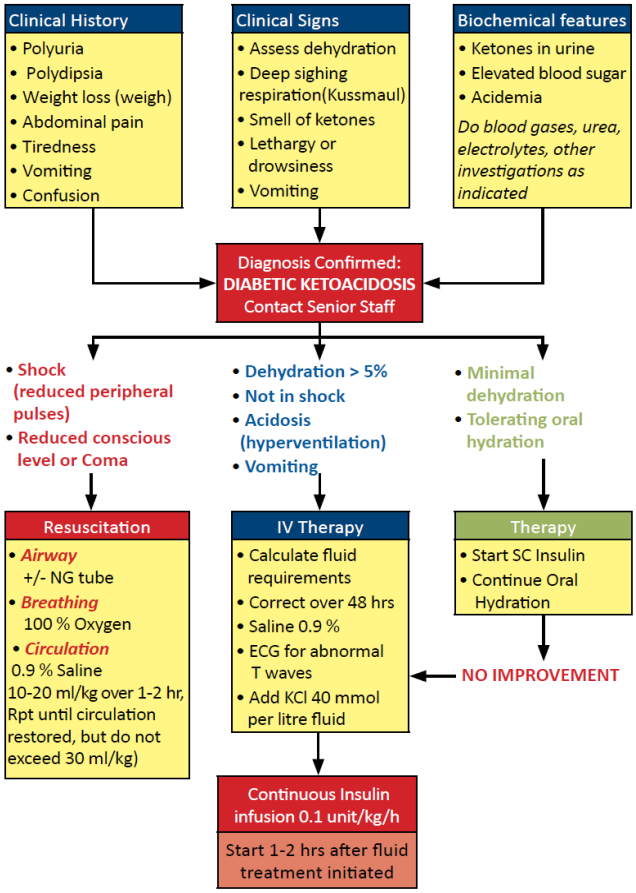
- Management
- General Objectives
- • Maintain normal glycemia with insulin therapy or oral medications (in type II diabetes mellitus) to prevent both the signs and symptoms of uncontrolled hyperglycemia and the complications mentioned above.
- Non-Pharmaceutical Management
- • Assess A-B-C-d (Airway, Breathing, Circulation, drug)
- • If patient has signs or symptoms of diabetic ketoacidosis (dKA) or hyperosmolar hyperglycaemic state, this is an emergency and treatment must be initiated immediately
- • The patient and the family should be counselled on the cause and treatment of diabetes and its management. The patient and the family should be taught how to monitor blood glucose, record the

test results, administer and adjust insulin doses based on blood glucose values and food intake.

- • They family should be counselled on the complications of diabetes mellitus and how to manage them. In particular, they should know the signs and symptoms of acute hypoglycemia and its management. They should also understand the importance of maintaining normoglycemia to avoid long-term complications. They should be instructed on how to manage acute illnesses in the context of diabetes mellitus, for example how to manage an insulin
- dose if the the patient is unable to tolerate oral intake.
- • diet modification is important in both type I and type II
- diabetes mellitus. A nutritionist should be involved in providing individualized recommendations.
- Pharmaceutical management
- • The majority of children with diabetes mellitus have type I diabetes and may present with diabetic ketoacidosis
- (dKA). The management of dKA is detailed below.
- → diabetes Mellitus Type I: Children with diabetes Mellitus Type I require insulin therapy. The patient is
- insulin dependent and while the insulin therapy may be adjusted based on the clinical condition and blood
- glucose results; the insulin therapy should NEVER be stopped completely as this could result in the
- development of dKA and death.

- **5.2. Diabetic Ketoacidosis**

Algorithm for Assessment and Management of Diabetic Ketoacidosis



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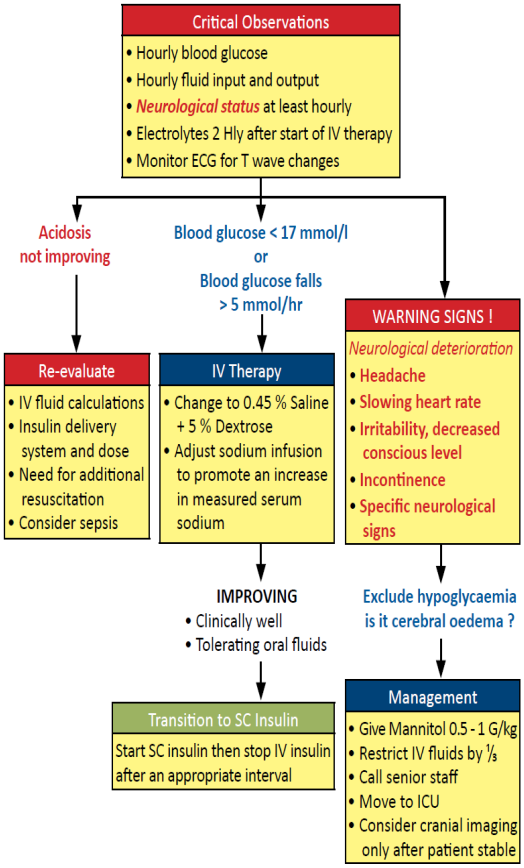
Algorithm for Assessment and Management of Diabetic Ketoacidosis *(cont)*



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Clinical and biochemical monitoring

- Monitoring should include the following:
  - Hourly (or more frequently as indicated) vital signs (heart rate, respiratory rate, blood pressure), head chart, accurate fluid I/O (including all oral fluid).
  - Amount of administered insulin.
  - Hourly capillary blood glucose (must be cross checked against laboratory venous glucose).
  - 2-4 hourly (or more frequent in more severe cases): BUSE, glucose, calcium, magnesium, phosphorus, hematocrit and blood gases.
  - 2 hourly urine ketones until cleared or blood b-hydroxybutyrate (BOHB) concentrations (if available).

Calculations
• Anion gap = (Na + K) - (Cl + HCO_3)
• Normal value: 12 +/- 2 mmol/L
• In DKA the anion gap is typically 20–30 mmol/L
• An anion gap > 35 mmol/L suggests concomitant lactic acidosis
• Corrected sodium (mmol/L) = measured Na + $\frac{2 \times (\text{plasma glucose} - 5.6)}{5.6}$
• Effective osmolality (mOsm/kg) = 2 x (Na + K) + plasma glucose + urea

Fluids and Salt

Principles of water and salt replacement

- Begin with fluid replacement before insulin therapy.
- Fluid bolus (resuscitation) required ONLY if needed to restore peripheral circulation.
- Subsequent fluid administration (including oral fluids) should rehydrate evenly over 48 hrs at a rate rarely in excess of 1.5 - 2 times the usual daily maintenance.

Acute Resuscitation

- If child is in shock, fluid resuscitation is needed to restore peripheral circulation, fluid boluses 10–20 mL/kg *over 1–2 hrs* of 0.9% saline is used.
- Boluses may be repeated, if necessary.
- There is no evidence that the use of colloids is better.

Replacement of water and salt deficits

- Patients with DKA have a deficit in extracellular fluid (ECF) volume. Clinical estimates of the volume deficit are subjective and inaccurate; therefore in
  - Moderate DKA use 5–7% deficit.
  - Severe DKA use 7–10% dehydration.

- ***Rehydrate the patient evenly over 48 hours:***
  - As a guide fluid infused each day usually < 1.5 - 2 times daily maintenance.
  - IV or oral fluids given in another facility before assessment should be factored into calculation of deficit and repair.
- Replacement should begin with 0.9% saline or Ringer's lactate for at least 4 - 6 h. Thereafter, use a solution that has a tonicity equal to or greater than 0.45% saline with added potassium chloride.
- Urinary losses should not routinely be added to the calculation of replacement fluid, but may be necessary in rare circumstances.
- Calculate the corrected sodium (formula as above) and monitor changes:
  - As plasma glucose decreases after IV fluids and insulin, the serum sodium should increase: this does not indicate a worsening of the hypertonic state.
  - A failure of sodium levels to *rise* or a further decline in sodium levels with therapy may signal impending cerebral oedema.
  - The sodium content of the fluid may need to be increased if measured serum sodium is low and does not rise appropriately as the plasma glucose concentration falls.
  - The use of large amounts of 0.9% saline has been associated with the development of hyperchloraemic metabolic acidosis.

Insulin therapy

- ***DKA is caused by either relative or absolute insulin deficiency.***
- ***Start insulin infusion 1–2 h AFTER starting fluid replacement therapy***
- Correction of insulin deficiency
  - Dose: 0.1 unit/kg/h IV infusion. (one method is to dilute 50 units regular insulin in 50 ml normal saline, 1 unit = 1 ml).
  - ***An initial IV bolus of insulin is not necessary***, and may increase the risk of cerebral oedema and should not be given.
- ***The dose of insulin should usually remain at 0.1 unit/kg/h at least until resolution of DKA*** (evidenced by pH > 7.30, HCO_3 > 15 mmol/L and/or closure of the anion gap), which takes longer than normalization of blood glucose concentrations.
- If patient has a marked sensitivity to insulin (e.g. young children with DKA, patients with Hyperglycemic Hyperosmolar State (HHS), and older children with established diabetes), the dose may be decreased to 0.05 unit/kg/h, or less, provided that metabolic acidosis continues to resolve.
- During initial volume expansion the plasma glucose concentration falls steeply. After commencing insulin therapy, the plasma glucose concentration typically decreases at a rate of 2–5 mmol/L/h.
- To prevent an unduly rapid decrease in plasma glucose concentration and hypoglycemia, ***add 5% glucose to IV fluid (e.g., 5% glucose in 0.45% saline) when plasma glucose falls to 14–17 mmol/L, or sooner if rate of fall is rapid.***
  - It may be necessary to use 10% - 12.5% dextrose to prevent hypoglycemia while continuing to infuse insulin to correct the metabolic acidosis.
- If blood glucose falls very rapidly *(> 5 mmol/L/h)* after initial fluid expansion add glucose even before plasma glucose has decreased to *17 mmol/L.*

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- If biochemical parameters of DKA (pH, anion gap) do not improve, reassess the patient, review insulin therapy, and consider other possible causes of impaired response to insulin; e.g. infection, errors in insulin preparation.
- If continuous IV insulin is not possible, hourly / 2-hourly subcutaneous (SC) or IM administration of a short or rapid-acting insulin analog (insulin Lispro or insulin Aspart) is safe / effective. (do not use in patients with impaired peripheral circulation)
  - Initial dose SC: 0.3 unit/kg, followed 1 h later at SC 0.1 unit/kg every hour, or 0.15–0.20 units/kg every 2 hours.
  - If blood glucose falls to <14 mmol/L before DKA has resolved (pH still < 7.30), add 5% glucose and continue with insulin as above.
  - When DKA has resolved and blood glucose is < 14 mmol/L , reduce SC insulin to 0.05 unit/kg/h to keep blood glucose around 11 mmol/L.

Important
If the blood glucose concentration decreases too quickly or too low before DKA has resolved: • Increase the amount of glucose administered. • Do not decrease the insulin infusion.

Potassium replacement

- ***There is always a deficit of total body of potassium (3-6 mmol/kg) even with normal or high levels of serum potassium at presentation. Replacement therapy is therefore required.***
- If immediate serum potassium measurements are unavailable, an ECG may help to determine whether the child has hyper- or hypokalemia.
- Subsequent potassium replacement therapy should be based on serum potassium measurements.
- Potassium replacement should continue throughout IV fluid therapy
- Maximum recommended rate of IV potassium replacement is 0.5 mmol/kg/h.
- If hypokalemia persists despite maximum rate of potassium replacement, then the rate of insulin infusion can be reduced.

Serum potassium level	Action
Hypokalemic at presentation	Start potassium replacement at the time of initial volume expansion and before starting insulin therapy, at a concentration of 20 mmol/ L (0.75 g KCl per pint).
Normokalemia	Start replacing potassium after initial volume expansion and concurrent with starting insulin therapy. The starting potassium concentration in the infusate should be 40 mmol/L (1.5 g KCl/pint)
Hyperkalaemia (K+ > 5.5 mmol/L)	Defer potassium replacement therapy until urine output is documented.

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Phosphate

- Depletion of intracellular phosphate occurs in DKA
- Severe hypophosphatemia, with unexplained weakness, should be treated
- Potassium phosphate salts may be safely used as an alternative to or combined with potassium chloride or acetate, provided that careful monitoring of serum calcium is performed as administration of phosphate may induce hypocalcaemia.

Acidosis

- *Severe acidosis is reversible by fluid and insulin replacement.*
- *There is no evidence that bicarbonate is either necessary or safe in DKA.*
  Bicarbonate therapy may cause paradoxical CNS acidosis, hypokalaemia and increasing osmolality.
- Used only in selected patients:
  - Severe acidaemia (arterial pH <6.9) in whom decreased cardiac contractility and peripheral vasodilatation can further impair tissue perfusion.
  - Life-threatening hyperkalaemia.
  - Cautiously give 1 - 2 mmol/kg over 60 min.

Introduction of oral fluids and transition to SC insulin injections

- Oral fluids should be introduced only with substantial clinical improvement (mild acidosis/ketosis may still be present).
- When oral fluid is tolerated, IV fluid should be reduced.
- When ketoacidosis has resolved (pH > 7.3; HCO_3^- > 15mmmol/L), oral intake is tolerated, and the change to SC insulin is planned, the most convenient time to change to SC insulin is just before a mealtime.
  e.g. SC regular insulin 0.25 u/kg given before meals (pre-breakfast, pre-lunch, pre-dinner), SC intermediate insulin 0.25 u/kg before bedtime.
  Total insulin dose is about 1u/kg/day.
- To prevent rebound hyperglycemia, the first SC injection is given 30 min (with rapid acting insulin) or 1–2 h (with regular insulin) before stopping the insulin infusion to allow sufficient time for the insulin to be absorbed.
- The dose of soluble insulin is titrated against capillary blood glucose.
- Convert to long-term insulin regime when stabilized. Multiple dose injections 4 times per day are preferable to conventional (twice daily) injections.

Morbidity and mortality

- In national population studies, mortality rate from DKA in children is 0.15–0.30%.
- Cerebral oedema accounts for 60–90% of all DKA deaths
- 10% - 25% of survivors of cerebral edema have significant residual morbidity.
- Other rare causes of morbidity and mortality include: sepsis; hypokalemia, hyperkalemia, severe hypophosphataemia; hypoglycaemia; aspiration pneumonia; pulmonary oedema; adult respiratory distress syndrome (ARDS); rhabdomyolysis; acute renal failure and acute pancreatitis.

Cerebral oedema

- Clinically significant cerebral oedema usually develops 4 -12 h after treatment has started, but may occur before treatment or rarely, as late as 24 - 48 h later.

Diagnostic Criteria for Cerebral Oedema	
• Abnormal motor or verbal response to pain	
• Decorticate or decerebrate posture	
• Cranial nerve palsy (especially III, IV, and VI)	
• Abnormal neurogenic respiratory pattern (e.g., grunting, tachypnea, Cheyne-Stokes respiration, apneusis)	
Major Criteria	**Minor Criteria**
• Altered mentation / fluctuating level of consciousness.	• Vomiting
	• Headache
• Sustained HR deceleration (decrease > 20 bpm), not attributable to improved intravascular volume or sleep state.	• Lethargy, not easily arousable
	• Diastolic blood pressure > 90 mmHg
• Age-inappropriate incontinence	• Age < 5 years

Treatment of cerebral oedema

- Initiate treatment as soon as the condition is suspected. (Mannitol and hypertonic saline should be available at the bedside)
- Give mannitol 0.5 - 1 g/kg IV over 20 min and repeat if there is no initial response in 30 minutes to 2 hours.
- Reduce the rate of fluid administration by one-third.
- Hypertonic saline (3%), 5 - 10 ml/kg over 30 min, may be an alternative to mannitol, especially if there is no initial response to mannitol.
- Elevate the head of the bed.
- Intubation may be necessary for the patient with impending respiratory failure. Maintain normocapnia. ($PaCO_2$ within normal range).
- After treatment for cerebral oedema has been started, a cranial CT scan should be done to rule out other possible intracerebral causes of neurologic deterioration.

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- 5.3. Hypoglycemia

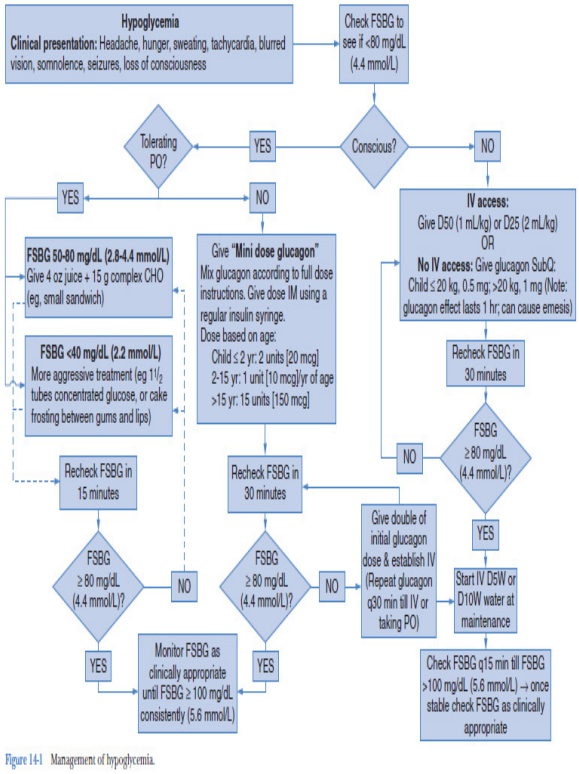

Figure 14-1 Management of hypoglycemia.

-
- Definition: Blood glucose levels below the lower limit of the norma
- range (blood glucose < 2.2 mmol/L, for malnourished children <mmmol/L).
- Causes/Risk factors
- - Individuals with diabetes
- - Excessive dose of medication anti–diabetic medication
- - Omitted or inadequate amount of food
- - Unaccustomed physical over activity
- - Alcohol intake
- Signs and Symptoms
- - dizziness
- - Blurred vision
- - Headaches
- - Palpitation
- - irritability and abnormal behavior
- - Sweating
- - Tremors
- - Tachycardia
- - Confusion

- - Unconsciousness
- - Convulsions
- Investigation
- - Blood glucose
- Management
- - 10% Glucose, IV, 2–4 ml/kg body weight 1 to 3 minutes through a large vein followed by 5–10% Glucose, IV, according to total daily fluid requirement until the patient is able to eat normally Alternatively
- • Glucagon, IV, IM or subcutaneous,
- • Age over 8 years (or body weight over 25 kg);→ Give 1 mg stat IM if available
- • Age less than 8 years (or body weight less than 25 kg);→ Give 500 microgram stat IM if available
- Recommendation
- - Control blood glucose 30 minutes after 10% bolus of Glucose

THYROID DISORDERS

Hypothyroidism

Congenital

• See chapter 34.

• **Acquired:** Prevalence is one in 500 to 1000 school-aged children)

• **Etiology**

• **Primary:** Autoimmune lymphocytic thyroiditis (Hashimoto's thyroiditis)**;** thyroidectomy or radioiodine therapy**;** irradiation to thyroid; iodide defi ciency; iodide excess (amiodarone),other goitrogens (lithium, cobalt)

• **Secondary:** Thyrotropin (TSH) defi ciency (isolated or associated with other anterior pituitary hormone defi ciencies); thyrotropin-releasing hormone defi ciency (hypothalamicinjury or disease)**;** large hemangiomas (consumption hypothyroidism); idiopathic

• **Clinical features:** Growth deceleration, mild weight gain, delayed puberty, constipation, cold
intolerance, delayed dental development, mental depression, metromenorrhagia, galactorrhea,precocious or delayed puberty; dry skin, brittle hair, hypotonia, hypothermia, bradycardia,transient deafness, delayed bone age

• **Evaluation**

T4 or FT4 with TSH in primary and decreased to normal TSH with T4 (or TSH elevation less than expected relative to degree of hypothyroxinemia) in secondary hypothyroidism

• Check anti-thyroid peroxidase and anti-thyroglobulin antibodies

• Evaluate for any other underlying cause

• Management

- • Levothyroxine (75–100 mcg/m2/d)

• Initially follow TSH and FT4; later TSH and T4 are used for monitoring effectiveness and adjusting the dose over time (if T4 and FT4 correlate)

• Follow up: If etiology is autoimmune (especially if multiple family members with autoimmune hypothyroidism) evaluate for other autoimmune disease

Hyperthyroidism

Acquired Hyperthyroidism

• Etiology: Graves disease, subacute thyroiditis, toxic phase of chronic lymphocytic thyroiditis,TSH-secreting adenoma, toxic multinodular goiter, factitious hyperthyroidism

• Clinical features: Hyperactivity, poor concentration, nervousness, emotional lability, fatigue,weight loss, increased sweating, heat intolerance, diarrhea, irregular menses, fi ne tremors, goiter,exophthalmos, palpitations, tachycardia, systolic hypertension, proximal muscle weakness

• Evaluation

• TSH (except in TSH-secreting adenoma where TSH); T3, T4, or FT4

• Check thyroid-stimulating immunoglobulin (TSI)

• Thyroid uptake scan (in Graves disease)

• Management

• ®**-adrenergic antagonists:** For control of symptoms (nervousness, tremors, tachycardia,and hypertension). ®-1 specifi c agents preferred (eg, atenolol)

• **Antithyroid agents (methimazole, propylthiouracil):** Interfere with thyroid hormone synthesis. PTU also T4 T3 conversion.

• **Radiation therapy:** An appropriate amount of oral I131 in children with Graves disease causes thyroid ablation. More commonly used as fi rst line treatment or for patients who are unresponsive or poorly adherent to antithyroid agents. Give ®-adrenergic antagonists during radiation therapy as symptoms worsen with cell lysis ~1-2 wk after ablation. Radiation therapyusually results in hypothyroidism, requiring future thyroid hormone replacement.

• **Surgical treatment:** Rare; indications include very large goiter, suspicious nodule,patients refusing radiation therapy, condition uncontrollable on methimazole or severely thyrotoxic patient requiring immediate intervention.

• **Iodide:** Only for acute management of severely thyrotoxic patient; large dose causes short-lived blockade of thyroid hormone synthesis and release. Continued use will cause a worsening of hyperthyroidism.

Thyroid Storm (rare but potentially lethal)

• **Clinical features:** In a patient with hyperthyroidism, acute onset of hyperthermia,tachycardia, restlessness; may progress to delirium, coma, or death if untreated.

• **Management:** Propanolol (or esmolol), propylthiouracil (PTU), iodine, steroids, cooling.

Other Thyroid Diseases

Acute thyroiditis

• **I ncidence/etiology:** Rare; common causes *GABHS, S. pneumoniae, S. aureus* and anaerobes.

• **Clinical findings:** The patient is always toxic with fever and chills. The thyroid gland is large, erythematous, and very tender ± fl uctuance. May have hoarseness or dysphagia.

TFT are usually normal.

• **Treatment:** Specifi c antibiotic therapy should be administered and surgical drainage performed if an abscess is present.

Subacute thyroiditis (de Quervain thyroiditis)

• **Incidence/etiology:** Rare; common causes are viral infection with mumps, influenza,

echovirus, coxsackievirus, EBV, or adenovirus

• **Clinical findings:** Onset is generally insidious. Similar to acute thyroiditis, and thyroid is firm and enlarged. Sedimentation rate is elevated. May have TFT.

Nonthyroidal illness (sick euthyroid syndrome; low T3 syndrome)

• Most common cause of abnormal thyroid tests in hospitalized patients.

T3, T4, reverse T3.

• TSH is usually low-normal, but may rise during recovery phase.

- • Issue of thyroid hormone replacement controversial.

SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE (SIADH)

AND CEREBRAL SALT WASTING (CSW)

COMPARISON OF SYNDROME OF INAPPROPRIATE ANTIDIURETIC

- **HORMONE AND CEREBRAL SALT WASTING**

	SIADH	CSW*
Pathophysiology	Inappropriate ADH secretion despite normal or increased plasma volume, which results in hyponatremic euvolemia or hypervolemia	Increased urine Na loss, which leads to hyponatremic dehydration
Etiology	**CNS:** Tumor or injury, surgery, meningitis, encephalitis, subarachnoid hemorrhage, hydrocephalus **Lung:** Pneumonia, TB **Other:** HIV/AIDS, neoplasm, thoracic surgery, drug use	CNS injury, tumor, meningitis (eg, bacterial, TB), subarachnoid or intracerebral hemorrhage, stroke, craniosynostosis repair
Clinical manifestation	**Symptoms:** Headache, blurred vision, nausea and vomiting, muscle cramps, weakness, irritability, change in mental status **Signs:** Not dehydrated; normal skin turgor; normal or ↑ BP; ± edema, ↓ DTR, asymmetric pupils, low GCS, pseudobulbar palsy	**Symptoms:** Polyuria, nausea, vomiting, headache, weight loss, ↑ thirst, altered mental status, seizures if severe hyponatremia, coma **Signs:** Moderate to severe dehydration (dry mucus membranes, sunken eye, sunken fontanel), orthostatic hypotension and ↑ HR, delayed capillary refill
Diagnostic evaluation	Euvolemic or hypervolemic on exam ↓ Na (< 135 mmol/L), ↓ serum osmolality (<280 mOsm/kg), ↓ serum uric acid, ↑ urine osmolality, ↓ U_{Na}, ↓ urine volume	Hypovolemic on exam ↓ Na, ↑ serum osmolality, ↓ serum uric acid, ↓ urine osmolality, ↑ U_{Na}, ↑ urine volume
Management	Fluid restriction (start with <75% of maintenance, ie, 1000 mL/m²/d); if no improvement, decrease to 50%, and so on Demeclocyline or AVP receptor antagonist 3% hypertonic saline only if Na <120 meq/L and child seizing	Correct intravascular volume and provide sodium replacement with IVF

*Also known as Renal salt wasting (RSW)

DIABETES INSIPIDUS

	Central Diabetes Insipidus	Nephrogenic Diabetes Insipidus
Etiology	CNS injury, tumor, meningitis, subarachnoid hemorrhage, stroke, DIDMOAD (Wolfram) syndrome	Familial: X-linked recessive inheritance (V_2 receptor gene defect); AR inheritance (aquaporin 2 gene defect). Others: Hypercalcemia, hypokalemia, drugs (eg, lithium), chronic renal disease

(continued on next page)

	Central Diabetes Insipidus	Nephrogenic Diabetes Insipidus
Clinical manifestation	Polyuria, polydipsia, nocturia, hypernatremia, dehydration, seizure (if severe hypernatremia)	Dehydration; infants exhibit irritability, poor feeding, growth failure, vomiting
Diagnostic evaluation	Low urine osmolality (50–300 mOSm/L) High serum osmolality (>300 mOsm/L) Low urine specific gravity <1.010 Water deprivation test may be needed for definitive diagnosis Central DI responds to vasopressin	Water deprivation test may be needed for definitive diagnosis Nephrogenic DI is resistant to vasopressin
Management	Management of hypernatremia Vasopressin IV for acute DI, coma, post-surgery DDAVP: PO, subcutaneous, or intranasal	Provision of free water Salt-restricted diet Drugs: Thiazide diuretics, amiloride, indomethacin

-

SEXUAL DEVELOPMENT

Normal Sexual Development

• In girls, thelarche (breast development) starts at 8–13 yr; varies by ethnic group) pubarche
(pubic hair development) menarche (2.3 yr ± 1 SD from thelarche)
• In boys, gonadarche (testicular enlargement) starts at 9–14 yr pubarche (secondary sexual characteristics, eg, voice change, growth spurt, pubic hair) occurring approximately halfway through the process

Delayed Puberty

• Girls: No thelarche by age 13 yr, or no pubarche by age 14 yr, or no menarche by age
16 yr or >5 yr between thelarche and menarche.
• Boys: No gonadarche by age 14 yr, or no pubarche by age 15 yr, or >5 yr required tocomplete testicular enlargement
• Pubertal arrest = No progress in puberty over two years.

Precocious Puberty

• Pubertal development in girls before age 8 yr and in boys before age 9 yr (there is ethnic variation for thelarche: caucasian girls as early as age 7 yr and African American girls as early as age 6 yr but without other signs of development).

- **TANNER STAGES IN GIRLS AND BOYS**

	Girls		Boys
Breast	B1 : Prepubertal	Gonadal	G1 : Prepubertal <2.5 cm length
	B2 : Breast bud		G2: Testes >2.5 cm length or ≥4 cc vol
	B3: Breast elevation		G3: Testes >3-3.5 cm length
	B4: Areolar mound ("mound on a mound")		G4: Testes >3.5-4 cm length
	B5: Adult		G5: Adult testicular length >4 cm

(continued on next page)

	Girls		Boys
Pubic hair	PH1: Prepubertal	Pubic hair	PH1: Prepubertal
	PH2: Sparse hair on medial labia		PH2: At base of penis
	PH3: Coarse, curly hair, spread over mons pubis		PH3: Spread to mons pubis
	PH4: Increasing distribution over labia majora		PH4: Not on thighs
	PH5: Adult (on inner thighs)		PH5: On thighs

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6. Musculoskeletal Conditions

6.1. Septic Arthritis

- Definition: Septic arthritis is defined as an acute articular suppurative
- infection caused by pyogenic micro-organisms
- Causes
- Neonates S.aureus, Group B. Streptococci, E. coli, fungi
- Infants/children S.aureus, H. influenzae, Group A Streptococci,S. pneumonia
- Children - sexually active N. gonorrhoea
- Chronic septic arthritis Brucella, tuberculosis, atypical mycobacteria, fungi and other uncommon organisms
- Risk factors
- - Trauma
- - Rheumatoid arthritis or osteoarthritis
- - Sickle cell disease
- - Skin infections
- - Sexual activity

- - Immune deficiency (HIV, etc.)
- signs and Symptoms
- - In neonates and infants
- • Signs and symptoms may be subtle (not well remarked)
- • diminished movement of the extremity
- • digestive disturbance
- • Poor progression of weight
- • Fever
- • Septicemia
- • Swollen, warm and painful joints
- - Older infants and children
- • Acute onset of pain, warm, and swollen joints
- • Usually monarticular and affecting large weight-bearing joints (knee, shoulder or hip)
- Complications
- - Sepsis
- - Osteomyelitis
- - destruction of articular cartilage, permanently damaging the joint Secondary infectious site (bacterial endocarditis, brain abscess, etc.)
- Investigations
- - Joint ultrasonography
- - Arthrocentesis with synovial fluid examination
- - FBC and CRP
- - X-ray
- - Blood culture and sensitivity before starting antibiotic treatment
- - Scintigraphy
- - MRI
- Management
- Non-pharmacological management
- • Emergency surgical drainage of pus from infected joints
- Pharmacological management
- • Antibiotics: minimum duration of therapy is 4–6 weeks
- → Neonates
- □ cloxacillin IV
- o 1st -2nd week of life: 50 mg/kg/dose every 12 hours
- o 3rd – 4th week of life: 50mg/kg/dose every 8 hours
- o > 4 weeks of life 50mg/kg/dose 6 hourly +
- Cefotaxime, IV, 50 mg/kg/dose (preterm 12 hourly, 1st week of life 8 hourly and > 2 weeks every 6 hours)

- → Infants and children
- □ cloxacillin IV 50mg/kg/dose, every 6 hours +
- Cefotaxime IV 25–50mg/kg/dose, every 6 hours
- □ do arthrocentesis and culture to treat appropriately to sensitivities
- □ Alternative: Vancomycine 50mg/kg/day divided in 3 doses. Maximum dose is 1g/dose
- Antipyretics and anti-inflammatories
- • Ibuprofen, oral, 5–10 mg/kg/dose, every 6 hours
- Recommendations
- - Penicillin antibiotic given for up to 6 weeks, with the first 2 weeks administered intravenously followed by a switch to oral treatment if an oral option exists and clinical signs, symptoms, and inflammatory markers are settling
- - IV antibiotics regimen is adjusted based on the results of culture and sensitivity testing
- **6.2. Juvenile Rheumatoid Arthritis**
- Definition:Juvenile Rheumatoid Arthritis is a chronic non-suppurative inflammatory condition of the synovium.
- OCCURS IN DIFFERENT FORMS
- - Systemic onset arthritis (Still's disease), occurs at any age (mostly between 2–4 years old)
- - Polyarticular onset arthritis, typically involves five or more joints, usually small joints Pauci – Articular onset Arthritis, most common type of juvenile rheumatoid arthritis (50 %), less than five joints affected
- SYSTEMIC ONSET ARTHRITIS

Figure 12–7 A 3-year-old boy with systemic JRA presenting with spiking fever and an exanthematous rash, eosinophilia, hepatomegaly, and adenopathy.

Signs and Symptoms

<< Back

- - Swinging/spiking fever
- - Rash – maculo–papular, especially on the torso
- - Lymphadenopathy
- - Hepato–splenomegaly
- - Arthralgia
- - Arthritis, multiple joints
- - Serositis, i.e. pericarditis and pleuritis
- POLYARTICULAR ONSET ARTHRITIS
- Signs and Symptoms
- - Affects ≥ 5 joints in the first 6 months
- - Involves large and small joints
- - Rheumatoid factor either positive or negative
- - Aggressive form of diseases with chronic course persisting into adulthood
- PAUCI – ARTICULAR ONSET ARTHRITIS

- 

Figure 12–8 Pauciarticular JRA.

- Signs and Symptoms
- - Involves the large joints (wrists, knees, ankles or elbows)
- - Often asymmetrical distribution
- - ≤ 4 joints are involved
- - Associated with an increased risk of iridocyclitis/uveitis
- Complications
- - Leg length discrepancy
- - Scoliosis
- - Contractures
- - Iridocyclitis/uveitis
- Investigations
- - FBC, differential, ESR
- - Rheumatoid factor
- - X–ray of affected joints
- - Anti Nuclear Antibodies (ANA)
- Management
- Non-pharmaceutical management
- • Occupational and physiotherapy are essential
- • Education of the patient and their families
- Pharmaceutical management
- • First Choice: Brufen 5-10 mg/kg/dose x 3/day
- • Alternative: Prednisone PO 2 mg/kg as a single daily dose for 1–2 weeks, continue with 0.3–0.5 mg/kg/day as single dose for 3 months
- If Arthritis not controlled
- • Give Methotrexate PO, 0.3 mg/kg/week as a single dose on an empty stomach, increase at monthly intervals up to 1 mg/kg/week until there is satisfactory response, maximum dose is 25 mg/week + folic acid 5mg daily for methotrexate treatment.
- Recommendation

- - Refer patient for rheumatology specialist consultation and adequate management (methotrexate treatment).

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7. Central Nervous System

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- **7.1. Epilepsy**
- Definition: Epilepsy is a condition characterized by recurrent seizures
- associated with abnormal paroxysmal neuronal discharges. When seizures are recurrent, persistent or associated with a syndrome, then the child may be diagnosed with epilepsy.
- Causes
- - Idiopathic (70-80%)
- - Secondary causes:
- • Cerebral dysgenesis or malformation
- • Cerebral vascular occlusion
- • Cerebral damage like Hypoxic Ischemic Encephalopathy
- (HIE), intraventicular hemorrhage or ischemia, head injury, infections
- • Cerebral tumors
- • Neuro-degenerative disorders
- Signs and Symptoms
-

Type	Clinical Signs/Symptoms
Infantile spasms (West's Syndrome)	- Onset is during the child's first year - Epileptic spasms (flexion and extension) associated with hypsarrhythmia on the EEG - Developmental regression - Child appears to stare, with a sudden flexion of the trunk and head, limbs either flung in or out but held in a tonic spasm for a few seconds - Red appearance in the face and may cry out
Severe Myoclonic Epilepsy of Infancy (SMEI)	- Occurs in children under 1 year of age - Recurrent clusters of febrile convulsions, severe neuro-regression and other non-febrile seizures by 2 - 3 years of age

-
-
-

Type	Clinical Signs/Symptoms
Lennox-Gastaut syndrome (LGS)	- Onset between at 2 - 3 years of age - Combination of Generalized Tonic Clonic Seizures (GTCS), atypical absences, myoclonic seizures, atonic drop attacks - Occasionally complex partial seizures - Behavioral problems and neuro-regression
Benign rolandic epilepsy with centrotemporal spikes (BRECTS)	- Onset at ± 6–10 years (can occur before or after 6 years up to 10 years of age) - Sleep related events of hemi-facial clonic spasm - Inability to speak with retained awareness - Usually resolves by late adolescence
Primary generalized absence seizure of childhood (petit mal)	- Onset 4 - 6 years of age - Short spells of motor arrest of maximum 15 seconds duration with little or no associated movements and no post-ictal effect
Generalized epilepsy with febrile seizures	- Febrile convulsions which persist beyond 6 years of age - Often family history of febrile convulsions - Occasionally associated with afebrile convulsions

-

Note: *Infantile spasms, Severe Myoclonic Epilepsy of Infancy and Lennox-Gastaut syndrome are regarded as malignant forms of epilepsy and are associated with neuro-regression and behavioral problems.*

Complications

- Status Epilepticus
- Trauma secondary to loss of consciousness during seizures
- Mental retardation

Investigations

- EEG
- MRI of the brain
- CT scan of the brain

-
- Management
- Non Pharmaceutical
- • Acute management
- → Manage Airway-Breathing-Circulation-disability and
- continue to monitor throughout seizures
- → Place patient on side at 20 – 30° head up to prevent
- aspiration
- → Monitor heart rate, respiratory rate, blood pressure,
- oxygen saturation (SaO2), neurological status, fluid balance
- → Monitor laboratory values including blood glucose,
- electrolytes, blood gases, toxicology screen and if indicated anticonvulsant blood levels
- → Control fever with tepid sponging
- → Administer oxygen to maintain SaO2 of ≥ 95%
- → If unable to protect airway or poor ventilation,
- consider use of an oral airway, bag-mask ventilation and/or intubation
- → Admit to pediatric ward or to Intensive Care Unit if indicated
- • Long-term management
- → Minimize the impact of the epilepsy by obtaining
- complete seizure control to maximize child's full potential
- → Educate the patient and parent or caregiver about epilepsy and associated complications (i.e. learning
- difficulties)

- Pharmacological treatment
- • Children <1 month of age
- → Refer to neonatology protocols for management of convulsions
- • Children >1 month of age
- → Monotherapy is preferred but combination therapy
- may be necessary. Combination therapy should be initiated by or in close consultation with a pediatric
- specialist or neurologist. drug levels are rarely
- indicated unless there is concern about toxicity or compliance
- → For acute generalized tonic clonic seizures
- ■ Diazepam rectal 0.5 mg/kg once OR IV 0.2-0.3mg/kg once
- May be repeated every 5 minutes for a total of 3 doses, monitor airway and breathing closely with repeat dosing
- ■ Alternative Medication (in the absence of diazepam)
- o Lorazepam IV 0.05- 0.1 mg/kg once, may be repeated in 5 minutes for a total of 3 doses
- Or
- o clonazepam IV 0.1 -0.15 mg/kg loading dose by slow IV injection
- → For refractory status epilepticus
- ■ Midazolam IV 0.1-0.3 mg/kg bolus followed by a continuous infusion starting at 1 ug/kg/minute.
- The infusion can be titrated upwards every 5 minutes as needed.
- → If persistent seizure activity after benzodiazepines
- ■ Phenobarbital 15 mg/kg IV or by NG tube
- loading dose over 15minutes, may use a dextrose containing solution. If no response after 30
- minutes, may repeat a 7.5 -10 mg/kg IV loading dose.
- Or
- ■ Phenytoin 15-20 mg/kg IV infused over 30 minutes in a dextrose-free solution
- → If seizures persist after loading dose of either Phenobarbital or Phenytoin
- ■ Please consult a specialist physician regarding combination therapy and referral for specialized care. Phenytoin and Phenobarbital may be used together but vital signs must be monitored closely and patient should be referred as soon as possible.

- ■ Monitor for bradycardia, arrhythmias, and hypotension and pause the infusion if these signs occur and restart at 2/3 of the initial loading dose.

Selecting antiepileptic drugs according to seizure types		
Seizure type	**First line**	**Second line**
Focal Seizures		
	Carbamazepine Valproate	Lamotrigine, Topiramate, Levetiracetam, Clobazam, Phenytoin, Phenobarbitone
Generalized Seizures		
Tonic-clonic / clonic	Valproate	Lamotrigine, Topiramate, Clonazepam, Carbamazepine[1], Phenytoin[1], Phenobarbitone
Absence	Valproate	Lamotrigine, Levetiracetam
Atypical absences, Atonic, tonic	Valproate	Lamotrigine, Topiramate, Clonazepam, Phenytoin
Myoclonic	Valproate Clonazepam	Topiramate, Levetiracetam Clobazam, Lamotrigine[2], Phenobarbitone
Infantile Spasm	ACTH, Prednisolone, Vigabatrin[3]	Nitrazepam, Clonazepam, Valproate, Topiramate
Footnote: *1, May aggravate myoclonus/absence seizure in Idiopathic Generalised Epilepsy.* *2, May cause seizure aggravation in Dravet syndrome and JME.* *3, Especially for patients with Tuberous Sclerosis.*		

Antiepileptic drugs that aggravate selected seizure types	
Phenobarbitone	Absence seizures
Clonazepam	Causes Tonic status in Lennox-Gastaut syndrome
Carbamazepine	Absence, Myoclonic, Generalised tonic-clonic seizures
Lamotrigine	Dravet syndrome, Myoclonic seizures in Juvenile Myoclonic Epilepsy
Phenytoin	Absence, Myoclonic seizures
Vigabatrin	Myoclonic, Absence seizures

-

Side effects and serious toxicity of Antiepileptic Drugs		
Antiepileptic Drug	**Common side effects**	**Serious toxicity**
Carbamazepine	Drowsiness, dizziness, ataxia, diplopia, rashes	Steven-Johnson syndrome[1], agranulocytosis
Clobazam[2] Clonazepam	Drowsiness, hypotonia, salivary and bronchial hypersecretion, hyperactivity and aggression	
Lamotrigine	Dizziness, somnolence, insomnia, rash	Steven-Johnson syndrome
Levetiracetam	Somnolence, asthenia, dizziness, irritability, behavioural change	
Phenobarbitone	Behavioural disturbance, cognitive dysfunction, drowsiness, ataxia, rash	
Phenytoin	Ataxia, diplopia, dizziness, sedation, hirsutism, gum hypertrophy megaloblastic anemia	
Sodium valproate	Nausea, epigastric pain, tremor, alopecia, weight gain, hair loss, thrombocytopaenia	Hepatic toxicity (< 2 yrs age), pancreatitis, encephalopathy
Topiramate	weight loss, somnolence, mental slowing, word finding difficulty, hypohidrosis, renal calculi	
Vigabatrin	drowsiness, dizziness, mood changes, weight gain	Peripheral visual field constriction (tunnel vision)
*Footnote: 1, Steven-Johnson syndrome occurs more frequently in Chinese and Malay children who carry the HLA-B*1502 allele.* *2, Clobazam is less sedative than clonazepam*		

-
- Recommendations
- The following conditions require referral for specialized services:
- - All cases of suspected infantile spasms or myoclonic seizures.
- - If there is concern for a secondary cause of epilepsy requiring
- further evaluation (examples include brain tumors, tuberous
- sclerosis, brain abscess, cysticercosis, etc.). This is particularly
- true in partial seizures where there may be a focal neurological problem.
- - Seizures that are not controlled with first-line medication within 1 month.
- - Seizures associated with neuro-regression.

- - Mixed seizure types within one patient.

Febrile Seizures

-
- **Definition**

• Seizures occurring in association with fever in children between 3 months and 6 years of age, in whom there is no evidence of intracranial pathology or metabolic derangement.

quote a figure of 3-4% of children < 5 years experiencing febrile seizures.

Classification of Febrile Seizures	
Simple Febrile Seizures	**Complex Febrile Seizures**
• Duration < 15 minutes	• Duration > 15 minutes
• Generalised seizure.	• Focal features
• Does not recur during the febrile episode	• > 1 seizure during the febrile episode
	• Residual neurological deficit post-ictally, such as Todd's paralysis

-
- **Management**

• Not all children need hospital admission. The main reasons are: -
• To exclude intracranial pathology especially infection.
• Fear of recurrent seizures.
• To investigate and treat the cause of fever besides meningitis/encephalitis.
• To allay parental anxiety, especially if they are staying far from hospital.
• Investigations
• The need for blood counts, blood sugar, lumbar puncture, urinalysis, chest X-ray, blood culture etc, will depend on clinical assessment of the individual case.
• lumbar puncture

Must be done if:

- Any signs of intracranial infection.
- Prior antibiotic therapy.
- Persistent lethargy and not fully interactive 6 hours after the seizure.

S*trongly recommended if*

- Age < 12 months old.

- First complex febrile seizures.
- In district hospital without paediatrician.
- Parents have difficulty bringing in child again if deteriorates at home.
• Serum calcium and electrolytes are rarely necessary.
• EEG is not indicated even if multiple recurrences or complex febrile seizures.
• Parents should be counselled on the benign nature of the condition
• Control fever
• Avoid excessive clothing
• Use antipyretic e.g. syrup or rectal Paracetamol 15 mg/kg 6 hourly for patient's comfort, though this may not reduce the recurrence of seizures.
• Parents should also be advised on **First Aid Measures during a Seizure.**
• Rectal Diazepam
• Parents of children with high risk of recurrent febrile seizures should be supplied with Rectal Diazepam (dose : 0.5 mg/kg).
• They should be advised on how to administer it if the seizures lasts more than 5 minutes.
• Prevention of *recurrent* febrile seizures.
- Anticonvulsants are not recommended for prevention of recurrent febrile seizures because:
• The risks and potential side effects of medications outweigh the benefits
• No medication has been shown to prevent the future onset of epilepsy.
• Febrile seizures have an excellent outcome with no neurological deficit nor any effect on intelligence.

Risk factors for Recurrent Febrile Seizures
• Family history of Febrile seizures
• Age < 18 months
• Low degree of fever (< 40 °C) during first Febrile seizure.
• Brief duration (< 1 hr) between onset of fever and seizure.
** No risk factor < 15 % recurrence* *≥ 2 risk factors > 30 % recurrence* *≥ 3 risk factors > 60 % recurrence*
Risk factors for subsequent Epilepsy
• Neurodevelopmental abnormality
• Complex febrile seizures
• Family history of epilepsy
Prognosis in Febrile Seizures
Febrile seizures are benign events with excellent prognosis
• 3 - 4 % of population have Febrile seizures.
• 30 % recurrence after 1st attack.
• 48 % recurrence after 2nd attack.
• 2 - 7 % develop subsequent afebrile seizure or epilepsy.
• No evidence of permanent neurological deficits following Febrile seizures or even Febrile status epilepticus.

-

- **7.1.1. Convulsive Status Epilepticus**

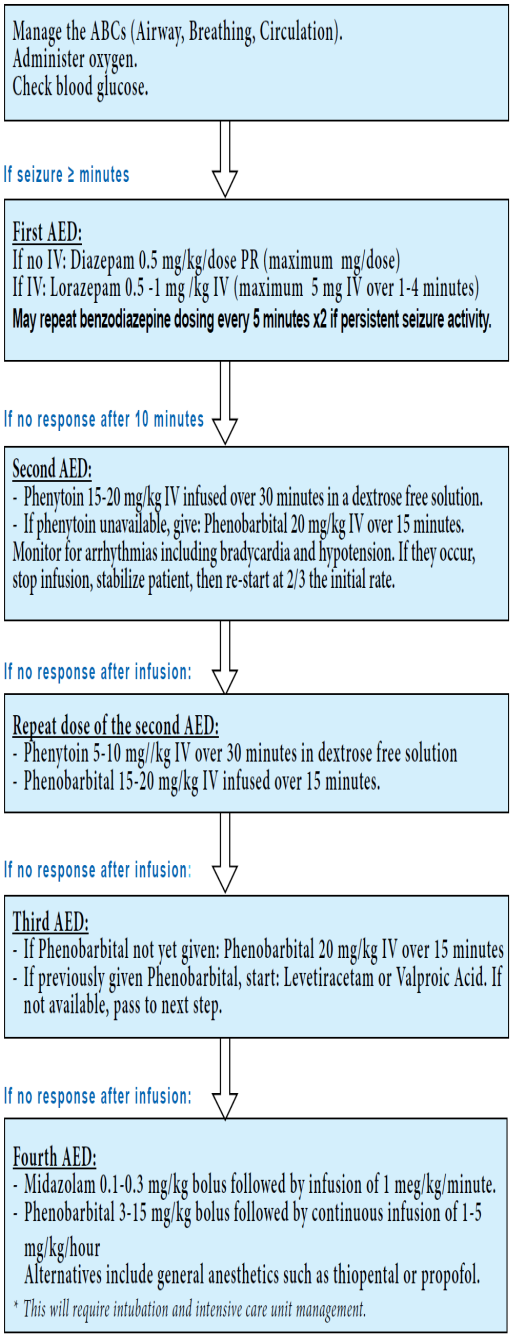
- Definition: Status epilepticus is a convulsion that persists for > 30
- minutes or is repeated frequently enough to prevent recovery of consciousness and return to baseline between attacks.
- Causes
- - Epilepsy syndromes may be present first as status epilepticus or status epilepticus may occur with inadequate anti-epileptic drug levels
- - CNS infection
- - Hypoxic ischemic insult
- - Traumatic brain injury
- - Cerebrovascular accidents

- - Metabolic disease including severe hypoglycemia and inborn errors of metabolism
- - Electrolyte imbalance
- - Intoxication
- - Cancer including primary brain tumors and metastatic disease
- Signs and Symptoms
- - Seizure lasting > 30 minutes or repetitive seizure activity without return to baseline consciousness.
- Complications
- - death
- - Neurologic morbidity including persistent seizures or Encephalopathy
- - Respiratory depression or failure due to neurologic status or aspiration
- - Blood Pressure disturbances including severe hypotension or severe hypertension
- - Hyperthermia
- - Metabolic derangement including hypoglycemia, alterations in sodium, and acidosis
- - Rhabdomyolysis
- - Renal failure
- Investigations
- - Laboratory evaluation for underlying cause may include blood glucose, electrolytes, NFS, arterial blood gas, toxicology screen,
- and anticonvulsant drug levels if indicated.
- - If there is no contraindication, a lumbar puncture should be performed to exclude infectious etiology.
- - EEG
- - CT scan of the brain
- - MRI of the brain
- Management
- Non-pharmaceutical Acute Management
- • Manage Airway-Breathing-Circulation-disability and continue to monitor throughout seizures
- • Place patient on side at 20 – 30° head up to prevent aspiration
- • Monitor heart rate, respiratory rate, Blood Pressure, oxygen
- saturation (SaO2), neurological status, fluid balance
- • Monitor laboratory values including blood glucose, electrolytes, blood gases, toxicology screen and if indicated anticonvulsant blood levels

- • Control fever with tepid sponging
- • Administer oxygen to maintain SaO2 of ≥ 95%
- • If unable to protect airway or poor ventilation, consider use of an oral airway, bag-mask ventilation and/or intubation
- • Admission to Intensive Care Unit if possible

Pharmacological

A flowchart showing medical management of Status Epilepticus:



-
- While following medication flow chart above, it is important
- to continue to address and manage the following:
- → ABCs

- → Hypoxia: Administer oxygen, oral airway, bag-mask
- ventilation or intubation.
- → Hemodynamic: Assess for shock or hypertension and
- manage accordingly.
- → Hyperthermia: Treat with Paracetamol 10-15 mg/kg
- orally or rectally every 4-6 hours as required.
- → Hypoglycemia: Treat with IV dextrose solution.
- → Hyponatremia: Assess etiology and manage
- accordingly.
- → If cerebral edema and normal renal function,
- consider Mannitol IV 0.5-1 gram/kg administered
- over 30–60 minutes.
- → If there is a known space-occupying lesion, consider
- Dexamethasone IV 1-2 mg/kg IV as a single dose
- then 1-1.5 mg/kg/day divided into 4 doses.
- Recommendations
- - Once status epilepticus is resolved, consider maintenance
- therapy with an appropriate anti-epileptic drug depending on the etiology of seizure.
- - Referral to a specialist is always appropriate in the case of status epilepticus. If possible, control seizures and stabilize the patient before referral. If status epilepticus has resolved, further work-up
- by a neurologist may be indicated.

- 7.2. Cerebral Palsy

- Definition: Cerebral palsy is a group of non-progressive clinical
- syndromes due to brain abnormalities from a variety of causes that is
- characterized by motor and postural dysfunction of varying severity.
- Though it is not progressive, the appearance of the brain lesions and the clinical manifestations may change over time as the brain matures.
- Causes
- - The etiology of the disorder is unknown in 70% of cases
- - Congenital infections (TORCH)
- - Obstetric complications (toxemia, placenta previa, abruptio placentae, etc.)
- - Teratogenic substances

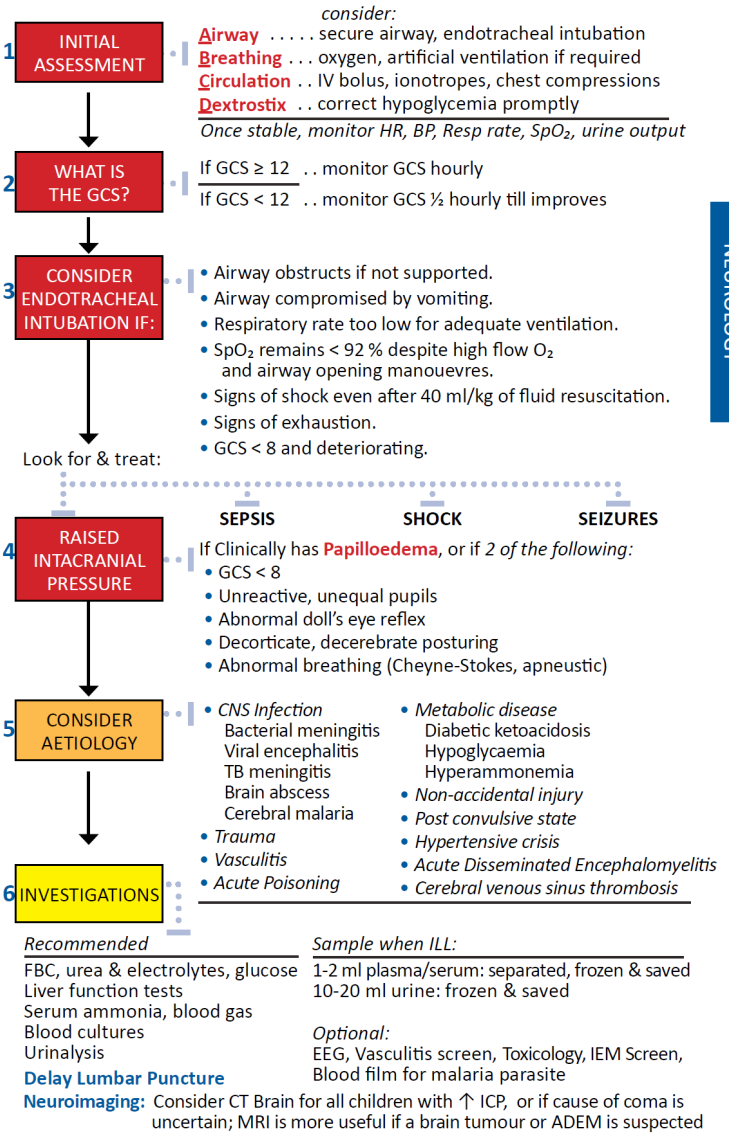
- - Congenital abnormalities including brain malformations and hereditary disorders Prematurity
- - Intracranial hemorrhage
- - Asphyxia: Please note that though this is often suspected as the cause, in reality perinatal asphyxia accounts for only a small percentage of cases
- - Cerebral trauma
- - Infections (Bacterial sepsis, meningitis, herpes)
- - Metabolic disturbances (kernicterus, severe prolonged hypoglycemia, Reye's syndrome)
- - Intoxication (i.e. lead)
- Signs and Symptoms
- Findings are consistent with a specific CNS lesion and commonly include:
- - Spastic syndromes : diplegia, hemiplegia, or quadriplegia
- - dyskinetic syndromes : athetosis, chorea or dystonia
- - Ataxic syndromes
- - Atonic syndromes
- - Abnormal persistence or absence of infantile reflexes
- Complications
- - Intellectual disability
- - Psychiatric disorders : Behavioral, emotional or psychiatric disorders
- - Epilepsy: This occurs in 45% of patients with CP and the onset is
- generally in the first 2 years of life
- - Speech, swallowing, vision and hearing problems
- - Growth failure: This is generally due to poor nutrition
- - Pulmonary disease: This is usually due to chronic aspiration and chronic pulmonary disease is a leading cause of death in patients
- with CP
- - Orthopedic disease: This includes hip and foot deformities and spinal curvatures. Patients may have chronic back, neck, and joint pain
- - Osteopenia: This is multifactorial related to poor nutrition, lack of motility and chronic medication use
- - Urinary disorders including enuresis, urgency, frequency and stress incontinence
- Investigations
- - Neuro-imaging including brain ultrasound, CT or MRI

- - Lumbar puncture if indicated
- - Basic lab-work to exclude other abnormalities (liver and renal function tests)
- - Genetic screening depending on clinical and family history
- - Metabolic screening depending on clinical and family history as well as basic lab work
- - EEG
- - Audiogram and visual evaluation to exclude correctable hearing or vision loss
- - X-rays if indicated
- Management
- - Perinatal asphyxia may be managed by passive or active hypothermia as per neonatology protocols.
- - Pharmacologic management of spasticity:
- • Botulinum toxin injections: Must be done by trained provider.
- • Dantrolene oral 0.5 mg/kg/dose once daily for 7 days, then increase to 1.5 mg/kg divided 3 times/day for 7 days, then increase to 3 mg/kg/day divided 3 times/day for 7 days, then increase to 6 mg/kg/day divided 3 times/day. do not exceed 400 mg/day.
- • Benzodiazepines: dose varies based on medication.
- Diazepam may be used: If 5 years: <8.5 kg: 0.5-1 mg at bedtime; 8.5-15 kg: 1-2 mg at bedtime; >5 years: 1.25 mg given 3 times per day up to 5 mg given 4 times per day.
- • Baclofen oral: <2 years: 10-20 mg divided every 3 times per day, titrate dose every 3 days in increments of 5-15 mg/day to a maximum of 40 mg daily; 2-7 years: 20-30 mg/day divided 3 times per day, titrate dose every 3 days in increments of 5-15 mg/day to a maximum of 60 mg/day, >8 years: 30-40 mg/day divided every 8 hours, titrate dose every 3 days in increments of 5-15 mg/day to a maximum of 120 mg/day.
- • Intrathecal Baclofen: Requires neurosurgical intervention to place pump to deliver medication. The benefits and
- complications should be discussed in detail with the neurosurgeon.
- - Multidisciplinary services to address and promote social and
- emotional development, communication, education, nutrition, mobility and maximal independence and normal appearance.
- • Physical, occupational, and speech language therapy as necessary
- • Social services provided in a variety of contexts to aid in the coordination of care.

- • Nutritional assessment and support for those with dysphagia and/or poor growth
- • Mobility aids including crutches, walkers, or wheelchairs as needed
- • Surgical procedures to correct spasticity, contractures, scoliosis, or hip disorders

-

Approach to The Child With Altered C



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7 MANAGEMENT

Management of Raised ICP

- Nursing
  - Elevate head up to 30°
  - Avoid unnecessary suction, procedures
- Fluid balance
  - Keep patient well hydrated
  - Avoid hypo-osmolar fluid, plain dextrose solutions
  - Care with sodium homeostasis:

↓serum sodium	→ ↓urine output	→ *consider* SIADH	→	*action* Fluid restriction
	→ ↑urine sodium	→ Cerebral salt wasting	→	Replace renal sodium losses

- Maintain cerebral blood flow
  - Keep CPP > 50 mmHg
  - If ↑ BP: do not lower unless hypertensive crisis, e.g. acute glomerulonephritis

Cerebral Perfusion Pressure (CPP)	=	Mean Arterial Pressure (MAP)	-	Intracranial Pressure (ICP)

- Use of IV Mannitol
  - Regular doses at 0.25 - 0.5 g/kg q.i.d. if required.
  - A CT scan to exclude intracranial bleeding is recommended.
- PaO_2 , $PaCO_2$ level
  - Maintain good oxygenation, normocapnia. i.e. $PaCO_2$ 4.0 - 4.6 kPa / 35 - 40 mmHg
- Surgical decompression
  - If medical measures fail, surgical decompression may be indicated (ie. external ventricular drainage, decompressive hemicraniectomy)

Treatment of Infection

- *Antibiotics*: In all children, unless alternative cause of coma is evident
- *Acyclovir*: In children with encephalitis, until CSF PCR results known
- *Others*: Anti-tuberculous therapy, anti-malarials

Treatment of Metabolic Encephalopathy

. . . . *refer section on* Metabolic disease in children

8 OUTCOME

General rules

- Outcome depends on the underlying cause: 1/3 die, 1/3 recover with deficits, 1/3 recover completely
- Acute complications improve with time. e.g. cortical blindness, motor deficits
- Metabolic causes may require long term dietary management.

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- **8. Dermatology**

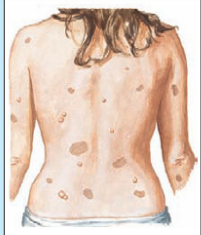
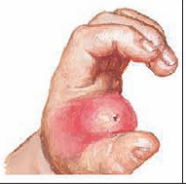
Type of primary lesion	Appearance	Description	Example	Type of primary lesion	Appearance	Description	Example
Macules		Flat changes in skin color of any size though generally less than 1 cm, may be rounded, irregular or fade into surrounding skin.	Café au lait macules, freckles, capillary malformations	Vesicle		Circumscribed, fluid filled elevation up to 1 cm in diameter	Coxsackie virus, Herpes simplex virus, varicella, miliaria crystallina
Patches		Greater than 1 cm flat lesion with color change, common colors include red (vascular lesion) darker (hyperpigmented) or lighter (hypopigmented or depigmented) than surrounding skin	Port wine stain, Mongolian spot, vitiligo	Bullae		Circumscribed, fluid-filled elevation greater than 1 cm in diameter. Flaccid bullae (superficial, involving the epidermis) rupture easily and intact lesions may not be evident. Tense bullae (subepidermal) remain intact.	Bullous impetigo, sucking blisters, Epidermolysis bullosa, blistering distal dactylitis, insect bite reaction
Papules		Solid elevations less than 1 cm, may have overlying color change or blend with surrounding skin	Molluscum contagiosum, dermal nevus, verruca/wart milia	Pustule		Less than 1 cm, circumscribed elevaton of the skin containing purulent material.	Folliculitis, transient pustular melanosis, infantile acropustulosis
Plaques		Elevated, flat-topped circumscribed lesion greater than 1 cm in diameter. May be formed by the confluence of papules.	Psoriasis, nevus sebaceous, lichen planus	Abscess		Circumscribed, elevated lesion greater than 1 cm containing purulent fluid.	Staphylococcal abscess, hidradenitis suppurativa, acne conglobata
Nodules or tumors		Circumscribed solid lesion less than 2 cm that involves the dermis and may include the subcutaneous tissue. Tumors are greater than 2 cm.	Dermoid cyst, Juvenille xanthogranuloma, neurofibroma, hemangiomas, lipoma	Wheal		An evanescent, elevated lesion that represents dermal edema. Lesions may vary in size and shape and are often surrounded by macular erythema.	Urticaria (hives), bug bite reaction (papular urticaria), urticarial vasculitis

Figure 122-2 *Primary lesions.*

- 8.1. Eczema

Figure 24.8 Atopic dermatitis. Inflamed skin worsened by rubbing/scratching. Itch is the key clinical feature in eczema at all ages, leading to an 'itch–scratch–itch' cycle.

Box 24.2 Some itchy rashes

- Atopic eczema
- Chickenpox
- Urticaria/allergic reactions
- Contact dermatitis
- Insect bites/papular urticaria
- Scabies
- Fungal infections
- Pityriasis rosea.

No itch? – then it's not eczema

Figure 122-3 *Flexural lichenification.*

Figure 122-5 *Nummular eczema.*

- Definition: Eczema, also known as dermatitis, is a syndrome characterized by superficial inflammation of the epidermis and itching.
- types
- - Atopic dermatitis: Chronic disease that affects the skin and often occurs together with asthma, dermatitis, rhinitis and conjunctivitis.
- - Contact dermatitis: Acute or chronic inflammation caused by allergens or irritants

- - Napkin (Or diaper area) dermatitis
- Signs and Symptoms
- - Pruritus (constant symptom)
- - And any of the following:
- • Blisters
- • Exudates and Erosions
- • Crusting/Excoriations
- • Xerosis
- • Erythroderma
- Complications
- - Secondary infection (bacterial, viral, fungal, etc)
- - Post inflammatory Hypo or Hyper pigmentation
- - Lichenification
- Investigations
- - Identification of allergens (Prick Skin Test or Patch test)
- - Full blood count (Increase of Eosinophiles)
- Management
- If Atopic dermatitis
- Non-pharmacological management
- • Patient education
- • Recommend Emollient to restore cutaneous barrier
- • Aqueous cream: Apply > 2 times/day
- • Emulsifying Ointment: apply > 2 times/day
- Pharmacological management
- • Local Treatment
- → Antiseptic – Exudative lesions, Potassium
- permanganate diluted at 1/10,000 (500mg Tablet in 5 liters)
- → Antibiotics – Impetiginized lesions, Fucidine 2% 1 application/day/5 days
- → Topical steroids
- ■ According to topography and thickness of the lesion
- ■ Short course of topical steroid treatment is recommended to avoid local side effects and gradual loss of efficiency
- →First choice
- ■ Clobetasol propionate (Dermovate) cream 2 applications/day for 3-4 days, then 1 application/day for 3 days then 1 application every 2 days/week for 2 weeks
- Or

■ Betamethasone dipropionate (Diprosone,
- Diprolene) cream/ointment 2 applications/day

- for 3-4 days, then 1 application/day for 3 days
- then 1 application every 2 days/week for 2 weeks
- → Alternatives: According to the severity of the lesions and location
- ■ Betamethasone valerate (Betneval) cream/
- ointment 2 applications/day for 3-4 days, then 1 application/day for 3 days then 1 application every 2 days/week for 2 weeks
- Or
- ■ Methylprednisolone (Advantan) cream/ointment 1 application/day/3-4days then every 2 days/week for 1 week
- Or
- ■ Hydrocortisone cream/ointment 2 applications/day for 3-4 days, then 1 application/day for 3days, then 1 application every 2 days/week for 2 weeks
- Note: Side effects of topical steroids:
- - Skin atrophy
- - Skin bleaching
- • Systemic treatment
- → Sedative antihistaminics: Promethazine Syrup: > 2 yrs of age 7.5-12.5ml at bed time until relief of scratching.
- → Combined Phototherapy UVAB in Erythrodermic atopic dermatitis
- Recommendations
- - Short duration of topical steroids whenever possible (stop topical steroids as soon as skin lesions disappear)
- - Encourage use of emollient
- - Avoid medicated soap
- - Other eczema, consider topical steroids as indicated in atopic
- dermatitis above

- 8.2. Bacterial Infections (Impetigo)

Figure 24.1 Bullous impetigo in a 2-week-old baby.

- Definition: A contagious intra-epidermal infection caused by
- streptococcus or staphylococcus and presenting as bullous lesions
- which rupture and crust. It comprises of two types namely:
- - non Bullous Impetigo: more common form and is a superficial infection of the skin that appears first as a discrete
- papullovesicular lesion surrounded by a localized area of redness.
- The vesicle becomes rapidly purulent and covered with crust. The lesions may occur anywhere but is more common on the face and extremities. There is usually neither fever nor systemic signs.
- Also occurs in traumatized skin that forms vesicles or pustules initially and rapidly develops crust.
- - Bullous Impetigo: less common and occurs most often in neonates and young infants on a previously healthy skin. It is characterized by transparent bullae usually < 3cm diameter. The distribution involves the face buttocks trunk and perineum. Staphylococcus aureus usually responsible.
- Signs and Symptoms
- - Non Bullous Impetigo
- - Honey colored crusters
- - Adenopathies
- - Bullous Impetigo
- - Flaccid and purulent bullous
- Complications
- - Ulcerations
- - Septicemia
- - Staphylococcal scaled skin syndrome (SSSS)
- Investigation
- - Swab for bacterial culture and sensitivity test
- Management
- - Local Treatment:
- • Antibiotics Fucidic acid ointment (Fucidine 2%) 2 applications/day/7 days
- • disinfectant with antiseptic solution:
- → Potassium Permanganate diluted at 1/10,000 (500mg in 5 liters)
- Or
- → chlohexidine solution (dermobacter) 2 applications/ day/7-10 days
- Systemic treatment-diffuse lesions
- • cloxacilline Syrup/Tabs 50mg/kg/day divided in 3 doses for 7 days

- Or
- • Erythromycine Syrup/Tab 50mg/kg/day divided in 3 doses for 7days
- Recommendation
- - Follow-up is important to ensure complete clearing of lesions
-

- 8.3. Fungal Infections

- 8.3.1. Dematophytes

Fig. 147 Tinea corporis (ringworm). A slowly spreading, scaly, erythematous lesion is present on the arm, due to a fungal infection. The spreading edge of the lesion is inflamed and the centre of the lesion is clearing giving a characteristic ringed appearance.

-

Figure 24.13 Ringworm of the scalp showing hair loss and kerion.

-
- Definition: Fungal infection often seen as Tinea or Ringworm with
- clinical entities/forms depending on the anatomic site and etiologic agents involved. It is of two types:

- - Tinea capitis: Fungal infection of the scalp or head and often
- found in children
- - Tinea corporis: Fungal infection of the glabrous skin (hairless part of the body)

Signs and Symptoms

TYPE	CLINICAL FORMS (Causative Agent)	SIGNS AND SYMPTOMS
Tinea Capitis	Microsporic Tinea *(Microsporum spp)*	- Large patches/ plaques - Hair fracture at few millimetres above surface of scalp (no alopecia)
	Tricophytic Tinea *(Tricophyton Spp)*	- Multiple small patches - Hair fracture at the scalp giving black dots aspect
	Inflammatory Tinea/ kerion *(Microsporum spp and Tricophyton Spp)*	- Severe inflammatory reaction with deep abscess causing hair loss with permanent alopecia after healing
		- Yellow cup shaped crusts known as scotula - Hair is eliminated leading to permanent alopecia
	Favus (*Tricophyton schonleini*)	- Raised borders with central normal skin, ring itself is red with dryness and scaling (circinate lesions)
Tinea Corporis	All *spp*	- Itching - Skin rash - Small area of red, raised spots and pimples - Rash which slowly becomes ring-shaped, with a red-colored, raised border and a clearer center - The border of rash may look scaly - Rash may occur on the arms, legs, face, or other exposed body areas-

-
- Investigations
- - Looking at a skin scraping of the rash under the microscope
- using a potassium hydroxide (KOH)test
- - Skin biopsy for histological exams
- Management

Types	Therapeutic options
Tinea capitis	- Topical treatment (always combined with systemic treatment) - Ketoconazol (Nizoral) shampooing, 3times/week apply to moist hair after shower / bath, and then wash off after 15 minutes ***Or*** **Systemic treatment** ***First choice*** - Whitefield ointment , apply BID - Griseofulvin (tabs 125mg,250mg, 500mg): 20 mg/ kg/ day, 6 to 8 weeks taken once daily with fatty meal ***Alternatives*** - Fluconazol (Flucazol susp 50mg/ml) 6 mg/kg/day, 6 to 8weeks once a day - If inflammatory Tinea: Add systemic antibiotics to antifungal as mentioned above
Tinea Corporis	**Local treatment** - Miconazole nitrate 2% cream, 2 applications/day for 15 days ***Or*** - Clotrimazol cream, 2 applications/ day for 10 days ***Or*** - Ketoconazole cream, 2 applications/ day for 10 days **Systemic treatment(≥3 lesions)** ***First choice*** - Griseofulvin 20 mg/kg/ day, 3-4 weeks taken with fatty meals ***Alternative*** - Fluconazol (Flucazole suspension, 50mg/ml) 6 mg/ kg/day, 6 to 8weeks once a day.

-
- Recommendation
- - Avoid sharing combs and towels to prevent Tinea capitis

- 8.4. Viral Infections

- 8.4.1. Herpes Zoster Virus (HZV) Infection

Figure 25-41

Figure 25-42

GENERAL EXAMINATION Lymphadenopathy Regional nodes draining the area are often enlarged and tender.

Sensory or Motor Nerve Changes Detectable by neurologic examination. Sensory defects (temperature, pain, touch) and (mild) motor paralysis, e.g., facial palsy.

Varicella-zoster virus infection: herpes zoster in T8 to T10 dermatomes Typical grouped vesicles and pustules with erythema and edema of three contiguous thoracic dermatomes on the posterior chest wall.

-
- Definition: It is a highly contagious systemic disease that normally results in lifelong immunity.
- Causes/Predisposing factors
- - Herpes zoster virus
- - People with no prior immunologic exposure to varicella virus, most commonly children, develop the clinical
- syndrome of varicella, while those with circulating varicella antibodies develop a localized recrudescence zoster (Zona)
- Signs and Symptoms
- - Small red macules that progress rapidly over 12 to 14 hours to papules
- - "dewdrops on a rose petal" Vesicles - pustules, and finally – crusts
- - Pruritus usually associated with skin lesions
- - Prolonged fever
- Complications
- - Bacterial super infection with subsequent scarring
- - Extra-cutaneous complication (CNS involvement, rare) with
- neurological manifestation
- - Hemorrhagic complications in immunocompromised children
- Management
- Immunocompetent children
- • Symptomatic therapy for non severe cases
- → calamine (ZnO + Fe2O3) lotion 4-5 application /day
- → Promethazine sp 5mg/5ml, 7.5mg at bed time > 2 -5 yr ; 12.5mg at bed time >6 yr (oral antihistaminic)
- • In severe cases (disseminated or mucosal involvement):

- → Acyclovir 20mg/kg a day for 5 days
- Immunocompetent ≥ 12 years
- • Symptomatic therapy in less severe disease
- → calamine (ZnO + Fe2O3) lotion 4-5 application a day
- → Oral antihistaminic: Promethazine 25mg at bed time associated with oral acyclovir 800 mg 5 times/day for 7 days
- Immunocompromised / Immunosuppressed children
- • Symptomatic therapy
- → calamine (ZnO + Fe2O3) lotion 4-5 application a day
- → Oral antihistaminic: Promethazine 25mg at bed time
- → Oral Acyclovir 800 mg 5 times/day for 7 days
- In life threatening conditions
- • Give IV Acyclovir: 10 mg/kg, infused at a constant rate over 1 h, every 8 hours for 7 days

- 8.5. Parasitic Infections

- 8.5.1. Scabies

:erion.

- **I.14** Scabies in a young child affecting
- Definition: Human scabies is a pruritic and contagious skin condition
- caused by the S. scabies mite var, hominis. It is transmitted via direct and
- prolonged contact with an infected individual.
- Sign and Symptoms
- - Nocturnal intense pruritus
- - Lesion distribution
- • Interdigital web spaces
- • Around the nipples

- • Genital region
- - Lesion characteristics
- • Papules, pustules or excoriations.
- • The pathognomonic sign: intradermal tunnel called scabietic "burrow"
- Complications
- - Secondary skin infection
- - Sepsis
- Investigation
- - Microscopic identification of skin scrapings
- Management
- - Benzyl Benzoate Emulsion (BBE) 25% (12.5% in children <5 yr, diluted in water 1:1, and 7.5% in infant, diluted in water 1:3), applied for 24 hours for three to five successive days. Apply from chin to toes and under fingernails and toenails. Repeat the same treatment ten days after.
- Or Permethrin 5% cream as follows:
- • Apply from chin to toes and under fingernails and toenails
- • Rinse off in shower / bath 12 hours later; repeat in 1 wk
- • Pediatric: >2 months old: Apply on head and neck, repeat in 1 wk + Promethazine sp5mg/5ml, 7.5mg/nocte > 2 -5 yr, 12.5mg nocte > 6 yr for 5 days
- Recommendations
- - All family members and close contacts must be evaluated and treated for scabies, even if they do not have symptoms
- - Instruct patients to launder clothing, bed linens, and towels used within the last week in hot water the day after treatment is initiated and again in 1 week
- - Items that cannot be washed may be professionally dry cleaned
- or sealed in plastic bags for 1 week

- 9. Infectious Diseases

- 9.1. Malaria

Uncomplicated Malaria

Symptomatic infection with malaria parasitaemia without signs of severity or evidence (clinical or laboratory) of vital organ dysfunction.

Treatment

UNCOMPLICATED PLASMODIUM FALCIPARUM

First Line Treatment	
Preferred Treatment	Alternative Treatment
Artesunate/Mefloquine (Artequine)#	Artemether/Lumefantrine (Riamet)+
Dosage according to body wt	*Dosage according to body wt*
10-20kg:* Artesunate 50mg OD x 3d Mefloquine 125mg OD x 3d (Artequine pellets)	5 -14 kg: D1: 1 tab stat then 1 tab again after 8 hours D2-3: 1 tab BD
20-40kg: Artesunate: 100mg OD x 3d Mefloquine 250mg OD x 3d (Artequine 300/750)	15 – 24kg: D1: 2 tabs stat then 2 tabs again after 8 hours D2-3: 2 tablets BD
>40kg: Artesunate 200mg OD x 3d Mefloquine 500mg OD x 3d (Artequine 600/1500)	25 – 35kg: D1: 3 tabs stat then 3 tabs again after 8 hours D2-3: 3 tablets BD
	>35kg: D1: 4 tabs stat then again 4 tabs after 8 hours D2-3: 4 tabs BD
Add primaquine 0.75mg/kg single dose OD if gametocyte is present at any time during treatment. ***Check G6PD before giving primaquine.***	
#. Avoid in children with epilepsy as well.	
*Use Riamet for children below 10 kg as there is no artequine formulations for this group of children.	
+ Riamet should be administered with high fat diet preferably to be taken with milk to enhance absorption.	
Both Artequine and Riamet are Artemisinin-based Combination Treatment (ACT)	

9.2. Meningitis

Fig. 142 Meningococcal septicaemia. A purpuric rash particularly affecting the limbs is present in this child with an acute febrile illness suggesting meningococcaemia. This may be followed by meningitis or associated with a more fulminating course, characterised by circulatory collapse and adrenal haemorrhage.

Meningococcal septicaemia

Figure 14.8 Rash of meningococcal infection. (**a**) Characteristic purpuric skin lesions, irregular in size and outline and with a necrotic centre. (**b**) The lesions may be extensive, when it is called '*purpura fulminans*'.

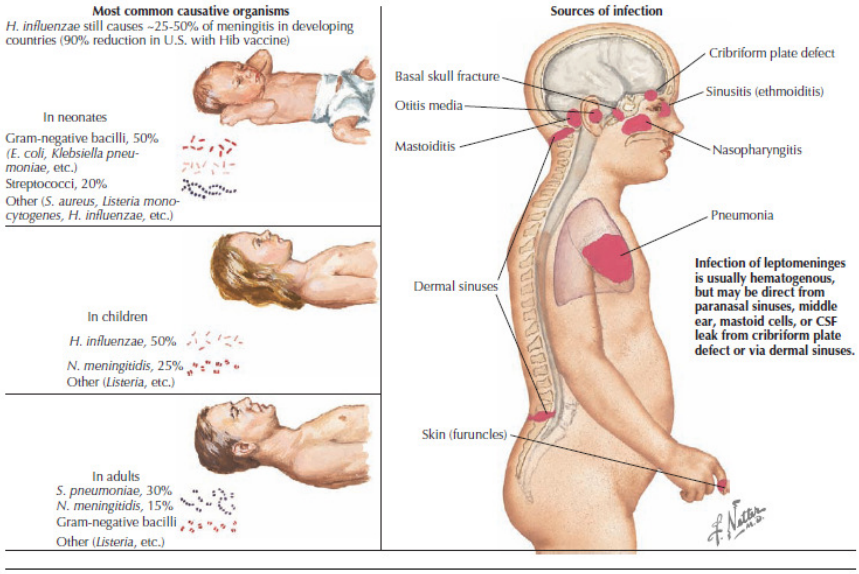
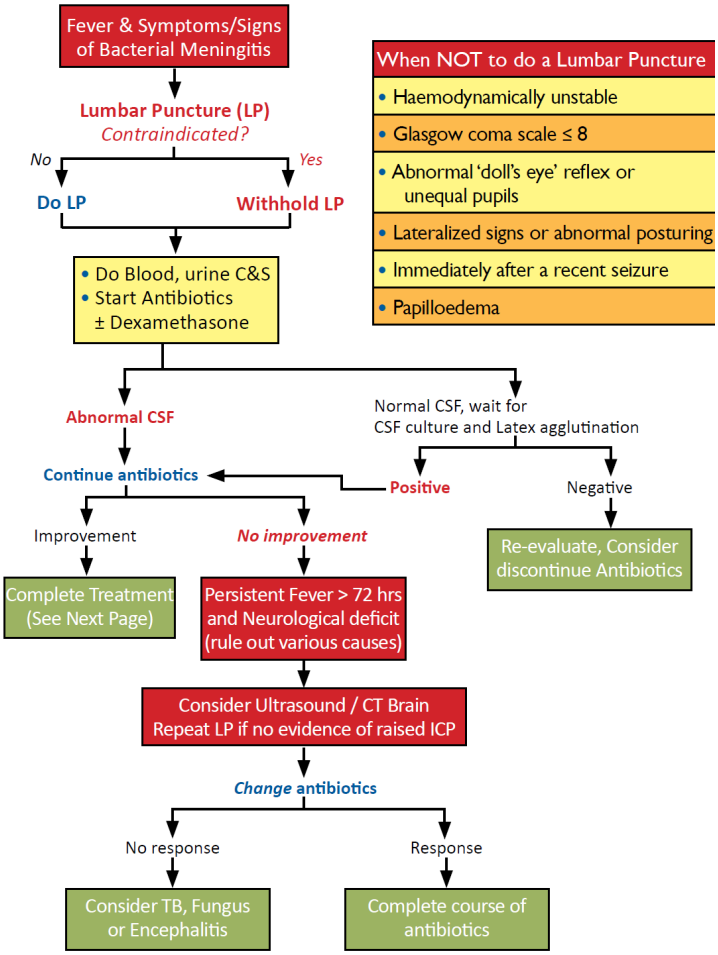


Figure 92-1 *Bacterial meningitis.*

Introduction

- Meningitis is still a major and sometimes fatal problem in Paediatrics.
- Morbidity is also high. A third of survivors have sequelae of their disease. However, these complications can be reduced if meningitis is treated early.

APPROACH TO A CHIILD WITH FEVER AND SIGNS/SYMPTOMS OF MENINGITIS



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Cerebrospinal fluid values in neurological disorders with fever				
Condition	Leukocytes (mm^3)	Protein (g/l)	Glucose (mmol/l)	Comments
Acute Bacterial Meningitis	100 - >50,000	Usually 1- 5	<0.5 - 1.5	Gram stain may be positive
Partially-treated Bacterial Meningitis	1 - 10,000 Usually high PMN, but may have lymphocytes	> 1	Low	CSF may be sterile in Pneumococcal, Meningococcal meningitis
Tuberculous Meningitis	10 - 500 Early PMN, later high lymphocytes	1- 5	0 - 2.0	Smear for AFB, TB PCR + in CSF; High ESR
Fungal Meningitis	50 – 500 Lymphocytes	0.5 - 2	Normal or low	CSF for Cryptococcal Ag
Encephalitis	10 - 1,000	Normal / 0.5-1	Normal	CSF virology and HSV DNA PCR

Recommended antibiotic therapy according to likely pathogen			
Age Group	Initial Antibiotic	Likely Organism	Duration (if uncomplicated)
< 1 month	C Penicillin + Cefotaxime	*Grp B Streptococcus* *E. coli*	21 days
1 - 3 months	C Penicillin + Cefotaxime	*Group B Streptococcus* *E. coli* *H. influenzae* *Strep. pneumoniae*	10 – 21 days
> 3 months	C Penicillin + Cefotaxime, OR Ceftriaxone	*H. influenzae* *Strep. pneumoniae* *N. meningitides*	7 – 10 days 10 – 14 days 7 days

Note:

- Review antibiotic choice when infective organism has been identified.
- Ceftriaxone gives more rapid CSF sterilisation as compared to Cefotaxime or Cefuroxime.
- If Streptococcal meningitis, request for MIC values of antibiotics.

MIC level	*Drug of choice:*
• *MIC < 0.1 mg/L (sensitive strain)*	*C Penicillin*
• *MIC 0.1-< 2 mg/L (relatively resistant)*	*Ceftriaxone or Cefotaxime*
• *MIC > 2 mg/L (resistant strain)*	*Vancomycin + Ceftriaxone or Cefotaxime*

4. Extend duration of treatment if complications e.g. subdural empyema, brain abscess.

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Use of Steroids to decrease the sequelae of bacterial meningitis

- Best effect achieved if given before or with the first antibiotic dose.
- Dose:
  Dexamethasone 0.15 mg/kg 6 hly for 4 days or 0.4 mg/kg 12 hly for 2 days
- Give steroids if CSF is turbid and patient has not received prior antibiotics.

Supportive measures

- Monitor temperature, pulse, BP and respiration 4 hourly and input/output.
- Nil by month if unconscious.
- Careful fluid balance required. Often, maintenance IV fluids is sufficient. However, if SIADH occurs, reduce to 2/3 maintenance for initial 24 hours. Patient may need more fluid if dehydrated.
- If fontanel is still open, note the head circumference daily. Consider cranial ultrasound or CT scan if effusion or hydrocephalus is suspected.
- Seizure chart.
- Daily Neurological assessment is essential.
- Observe for 24 hours after stopping therapy and if there is no complication, patient can be discharged.

If persistent fever in a patient on treatment for meningitis, consider:

- Thrombophlebitis and injection sites e.g. intramuscular abscess.
- Intercurrent infection e.g. pneumonia, UTI or nosocomial infection.
- Resistant organisms. Inappropriate antibiotics or inadequate dosage.
- Subdural effusion, empyema or brain abscess.
- Antibiotic fever.

Follow up (Long term follow up is important)

- Note development of child at home and in school.
- Note head circumference.
- Ask for any occurrence of fits or any behavioural abnormalities.
- Assess vision, hearing and speech.
- Request for early formal hearing assessment in cases of proven meningitis.
- Until child shown to have normal development (usually until 4 years old).

Prognosis depends on

- Age: worse in younger patients.
- Duration of illness prior to effective antibiotics treatment.
- Causative organism: more complications with *H. influenzae*, *S. pneumoniae.*
- Presence of focal signs.

Indications for Head CT Scan
Useful to detect complications
• Prolonged depression of consciousness
• Prolonged focal or late seizures
• Focal neurological abnormalities
• Enlarging head circumference
• Suspected subdural effusion or empyema

Indications for Subdural drainage
• Rapid increase in head circumference with no hydrocephalus
• Focal neurological signs
• Increased intracranial pressure
• Suspected subdural empyema

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- 9.3. Tetanus

- Definition: Tetanus is toxi infection of the nervous system with the
- potentially deadly bacteria clostridium tetani (C. tetani). It occurs in
- several clinical forms including generalized, neonatal and localized
- disease.
- Cause
- - Clostridia tetani
- Signs and Symptoms
- - Trismus (lock jaw)
- - Opisthotonos (Rigid arching of back mucles)

- - dysphagia
- - Laryngospasm
- - Autonomic nervous system instability with hypertension,
- tachycardia and dysarhythmias
- Complications
- - Asphyxia
- - Heart failure
- - Pneumonia
- - Fractures
- - Brain damage due to lack of oxygen during spasms
- Investigations
- - No specific lab test is available to determine the diagnosis of
- tetanus
- - Other tests done to rule out meningitis, rabies, strychnine
- poisoning etc.
- Management
- Non-Drug Treatment
- • Admit to High or Intensive Care Unit (if available)
- • Oxygen to prevent hypoxia and ventilatory support if needed
- • Monitor:
- → Temperature
- → Respiration
- → Heart rate
- → Blood gases
- → Sao2
- → Blood Pressure
- → Blood glucose
- → Electrolytes
- → Acid–base status
- • Protect the patient from all unnecessary sensory and other stimuli
- • Ensure adequate hydration and nutrition
- • Wound care and debridement/umbilical cord care
- • Educate parents/caregivers regarding prevention of tetanus by vaccination
- Pharmacological
- • Tetanus immunoglobulin, IM, 500–2 000 IU as a single dose
- • Eliminate toxin production
- → Benzylpenicillin (Penicillin G), IV, 50000IU/kg/day (Neonate every 12hours and in older children every 6 hours)

- → Metronidazole 40mg/kg/day IV in three divided doses for 7-10 days

	Weight	Dosage
Neonates less than 7 days old	<1.2 kg	7.5mg/kg/ i.v Every 48 hours
	1.2-2 kg	7.5kg/kg i.v Every 24 hrs
	> 2kg	15kg/kg/day Every 12 hours
Neonates 7 days and older	<1.2kg	7.5kg/kg Every 48 hours
	1.2-2 kg	15mg/kg/day Every 12 hours
	>2kg	30mg/kg/day Every 12 hourls
Infants and children		30mg/kg/24 i.v every 6 hourls

- → Diazepam, IV, 0.1–0.2 mg/kg/dose 4–6 hourly,
- titrated according to response. do not exceed dose
- of 10 mg/dose. Alternating with chlorpromazine 0.5 mg/kg every 6 hours PO (NGT)
- After recovery from tetanus, patients should be actively immunized as the disease does not confer immunity
- Note: Don't remove the nGT from the child until at least one week
- seizure free
- Prevention of tetanus
- - Minor Wounds
- • Children with clean minor wounds do not require tetanus immunoglobulin or antibiotics
- • Tetanus vaccine should be given, except in fully immunised patients who have received a booster within the past 5 years
- - For more severe wounds
- • If child with penetrating wound not completely immunised
- → Tetanus immunoglobulin, IM
- □ < 5 years 75 IU
- □ 5–10 years 125 IU
- □ > 10 years 250 IU
- → Tetanus Toxoid vaccine (TT), IM, 0.5 ml
- → Phenoxymethyl penicillin, oral, 12.5 mg/kg/dose every 6 hours for 7 days Or
- → Erythromycin, oral, 6.25–12.5 mg/kg/dose, every 6 hours for 7 days (f allergic to penicillins)
- Recommendation

- - Refer all severe cases of tetanus to Intensive Care Unit

- 9.4. Hepatitis

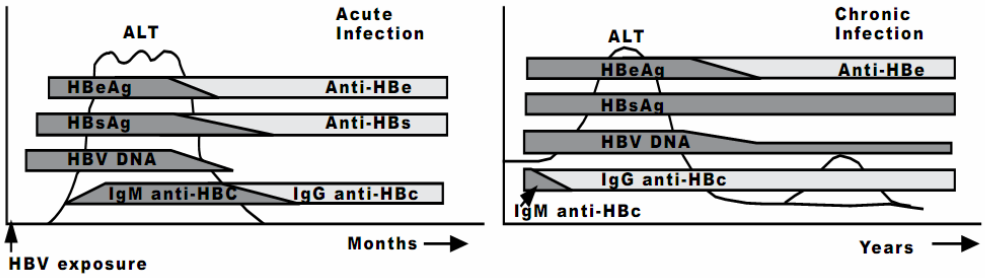
- Definition: It is an acute inflammation of the liver with varying degrees of hepatocellular necrosis. The most commonly known are hepatitis A, B and less commonly C, d and E viruses.
- HEPATITIS A
- Causes
- - Hepatis A RNA (virus)
- - Vaccination does exist but provided in developed countries
- - HAV is spread via the fecal-oral route
- Signs and Symptoms
- - Abrupt onset with nonspecific symptoms, such as fever, malaise,
- anorexia, vomiting, nausea, abdominal pain or discomfort, and diarrhea
- - Jaundice usually occurs one week after onset of symptoms, along with coliuria (bilirubin in the urine) and mild hepatomegaly
- - Several young children are asymptomatic. Symptomatic 30% of infected children who are younger than six years of age, jaundice usually lasts for less than two weeks. Conjugated bilirubin and aminotransferases returns to normal within two to three months
- - In contrast, older children and adults with HAV infection are usually symptomatic for several weeks. Approximately 70% are jaundiced, and 80% have hepatomegaly. Symptoms lasting for a longer time
- - The most common extrahepatic manifestations include an evanescent rash (11%) and arthralgias (14%). And less common
- extrahepatic manifestations include vasculitis, arthritis, optic neuritis, transverse myelitis, encephalitis, and bone marrow suppression
- Complications
- - Acute liver failure is rare in developed countries , but account for 60% of liver failure in Latin America
- - death
- Investigations
- - Liver Function tests
- - Anti-HAV IgM in a patient with the typical clinical presentation
- - Serological tests for Hepatitis A
- Management
- - Improved sanitary conditions, adherence to sanitary practices,

- hand washing +++ (virus may survive for up to four hours on the fingertips)
- - (Chlorination and certain disinfecting solutions are sufficient to inactivate the virus)
- - No specific treatment for Hepatitis A
- - Bed rest may be recommended
- - Active vaccine is recommended for all children 12- 24 months
- - Human immunoglobulin prophylaxis for those who had contact
- Patients rarely require hospitalization except for those who develop
- fulminant hepatic failure. The following criteria were proposed by the Pediatric Acute Liver Failure Study Group:
- • Absence of known chronic liver disease
- • Evidence of hepatic injury
- • PT>15 and/or INR>1.5 with encephalopathy
- • PT>20 and/or INR>2.0 with or without encephalopathy
- These criteria should be fulfilled within eight weeks from the onset of illness, and the above-described coagulopathy (prolonged prothrombin time and/or INR) should be unresponsive to vitamin
- K therapy. If suspicion refer to a specialist.
- HEPATITIS B
- Causes
- - Hepatitis B dNA virus (HBV)
- - Perinatal transmission remains the most important cause of chronic infection because of high rates of disease in pregnant women
- - Infants born to women with HBV infection (HBeAg positive or negative) shall be tested for hepatitis B at 9 – 18 months even if vaccinated (at least 5% develop chronic HBV)
- - Hepatitis B vaccination is part of national immunization program
- - All pregnant women should be screened for HBV infection
- Signs and Symptoms
- Infection with HBV is associated with characteristic changes in the serum levels of hepatitis B antigens and antibodies. These markers are used to define different clinical states
- Acute hepatitis
- • Acute HBV infection in children has a variable course ranging from asymptomatic infection to fulminant
- hepatitis.
- → Constitutional symptoms, anorexia, nausea, jaundice and right-upper-quadrant discomfort

- → The symptoms and jaundice generally disappear after one to three months, but some patients have
- prolonged fatigue even after normalization of serum aminotransferase concentrations. Older children and adolescents have mild constitutional symptoms
- during acute HBV infection
- Chronic hepatitis
- •Most children with chronic HBV infection are asymptomatic and grow and develop normally. Some
- children note vague right upper quadrant discomfort and fatigue, loss of appetite, occasional boots of mild jaundice.
- Chronic HBV infection is occasionally associated with extrahepatic manifestations including polyarteritis nodosa and glomerulonephropaty. The diagnosis of chronic HBV
- infection is based on persistence of HBsAg for more than six months; IgG anti-HBc is positive, while IgM anti-HBc is negative.
- Note: Some carriers have large numbers of HVB in their serum and liver without symptoms or signs and without antibodies in their serum.

Investigations

- Serologic responses to HBV infection:



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- - Left panel: Acute infection: HBeAg (hepatitis B e antigen),
- HBsAg (hepatitis B surface antigen), and HBV dNA beginning in the preclinical phase. IgM anti-HBc (hepatitis B core antigen) appears early in the clinical phase; the combination of this
- antibody and HBs Ag makes the diagnosis of acute infection.
- - Recovery: normalization of the serum ALT, the disappearance of HBV dNA, HBeAg to anti-HBe seroconversion, and subsequently HBsAg to anti-HBs seroconversion and switch from IgM to IgG

anti-HBc. Then previous HBV infection is characterized by anti-HBs and IgG anti-HBc.

- - Right panel: Chronic infection Persistence of HBsAg for more than six months after acute infection is considered indicative of
- chronic infection: persistence of HBeAg (for a variable period), HBsAg, and HBV dNA in the circulation; anti-HBs is not seen
- Other tests
- • Liver Function tests (Prothrombin time, Bleeding time)
- • Glycemia if severe
- • HBV tests (refer to figure)
- • Blood ammonia
- • Urea and electrolytes in cases of liver failure
- • CBC to determine severity of anaemia
- Complications
- - Chronic Liver disease: In children born from infected mothers approximately 76% of children remained HBeAg positive at 10 years of age. Rates of spontaneous seroconversion are less than
- 2% per year in children younger than three years of age, and 4 to 5% after age three. The frequency of spontaneous seroconversion increases during puberty (Cirrhosis)
- - Liver failure (hepatic encephalopathy) Portal hypertension (GIT bleeding, hematemesis and melena
- stools) Glomerulonephritis /Renal failure
- - Liver cancer
- Management
- - General measures
- • Counseling of the patient including alcohol use in adolescents and family, surveillance for disease progression
- and development of complications
- • Patients who are in the immune tolerant phase of HBV infection (ie, HBsAg positive, HBeAg positive, serum HBV
- dNA>20,000 copies/mL) should undergo monitoring of liver biochemical tests every 6 to 12 months
- • Patients who are in the inactive carrier phase of hepatitis B infection (ie, HBsAg positive, HBeAg negative, anti HBe positive, persistently normal ALT/AST levels, serum HBV
- dNA <10(5) copies/mL) should undergo monitoring of liver biochemical tests every 6 to 12 months
- - Selection of patients for treatment:
- • Treatment is generally considered in patients with HBV

- dNA positive chronic hepatitis who are in the immune active phase (usually defined as ALT/AST >2 times Upper Limits of Normal and HBV dNA >20,000 IU/mL or 10(5) copies/mL, for at least six months)
- • Children with ALT values greater than 10 times the upper limit of normal but with concomitant low HBV dNA levels may be in the process of spontaneous seroconversion, and may not require treatment. These patients should be observed for several months with serial serologic testing
- • If there is evidence of hepatic decompensating, such as jaundice or coagulopathy, treatment should be initiated earlier
- • Several other considerations may be relevant to treatment decisions (co-infected with HCV, HIV or HdV)
- - Choice of treatment
- • Lamivudine and interferon (IFn), are licensed for use in children
- • Adefovir approved for use in children over 12 years of age
- • Licensed in children with HIV and is a first choice for HBV in adult
- • Start using Lamivudine and TDF
- Diseases
- • Use IFN alfa as the first-line treatment (but expensive) for patients with serum ALT more than twice the upper limit of normal, have positive HBeAg, who are committed to adhering to the treatment, and have no comorbid diseases that might be exacerbated by an immunostimulatory agent
- • If the patient does not respond to IFN alfa (defined by detectable HBV dNA and elevated serum ALT six months after completion of the course of IFN alfa), a nucleoside/nucleotide analog such as Lamivudine or adefovir can be used – this shall be considered as primary treatment if IFN alpha is not available
- 9.5. Acute Liver Failure

Definitions

- *Fulminant hepatic failure* (HF): hepatic dysfunction (hepatic encephalopathy and coagulopathy) within 8 weeks of evidence of symptoms of liver disease and absence of pre-existing liver disease in any form.
- *Hyperacute/ Fulminant HF*: encephalopathy within 2 weeks of onset of jaundice.
- *Subfulminant HF:* encephalopathy within 2-12 weeks of onset of jaundice.
- *Subacute/ Late-onset HF*: encephalopathy later than 8 weeks to 6 months of onset of symptoms.

Salient features

- Jaundice with impalpable liver or a liver of reducing size.
- Encephalopathy - may worsen rapidly (needs frequent review).
- Bruising, petechiae or bleeding from deranged clotting unresponsive to vitamin K.
- Failure to maintain normoglycaemia (which aggravates encephalopathy) or presence of hyperammonaemia.
- Increased intracranial pressure (fixed dilated pupils, bradycardia, hypertension and papilloedema).

Grading of Hepatic Encephalopathy - Coma Level
Grade 1
Irritable, lethargic
Grade 2
Mood swings, aggression, photophobia,
Not recognising parents, presence of flap
Grade 3
Sleepy but rousable, incoherent, sluggish
Pupils, hypertonia ± clonus, extensor spasm
Grade 4
Comatose; decerebrate, decorticate
or no response to pain

Causes of Hepatic Failure
Infection
Hepatitis A, B, non A- non B, CMV
Leptospirosis, Dengue
Herpes simplex virus (particularly in small infants)
Drugs
Carbamazepine, valproate
Paracetamol, halothane
Ingested toxins
Mushrooms, Amanita phalloides
Metabolic
Fructosaemia, galactosaemia, tyrosaemia,
Wilson's disease
Neonatal haemochromatosis
Ischaemic shock
Gram negative septicaemia,
Budd Chiari syndrome
Autoimmune
Autoimmune Hepatitis
Tumour
Histiocytosis, lymphoproliferative disorder

Principles of management

Supportive Treatment

- Nurse in quiet darkened room with head-end elevated at 20° with no neck flexion (to decrease ICP and minimise cerebral irritability).
- DO NOT SEDATE unless already ventilated
  - This may precipitate respiratory failure and death.
- Maintain blood glucose between 6-9 mmol/l using *minimal fluid volume* (40-60 ml/kg/day crystalloid) with high dextrose concentrations e.g. 10-20%. Add Potassium as necessary.
- Check capillary blood sugar every 2 - 4 hourly.
- Strict monitoring of urine output and fluid balance. Catheterise if necessary.
- Check urinary electrolytes, serum urea, creatinine, electrolytes, osmolarity.
- Frequent neurological observations (1-4 hourly).
- Maintain oxygenation with facial oxygen.
- Give Vitamin K to correct prolonged PT. If frank bleeding (GIT/oral) occurs, consider prudent use of FFP or IV Cryoprecipitate at 10 ml/kg.
- Prophylactic Ranitidine + oral Antacid to prevent gastric/duodenal ulceration.
- Full septic screen (excluding LP) on admission, CXR. Treat sepsis aggressively, monitoring levels of aminoglycosides frequently.

- Stop oral protein initially. Gradually reintroduce 0.5-1g/kg/day.
- Lactulose to produce 3-4 loose stools per day.
- **Strict fluid balance is essential* - aim for urine output > 0.5 ml/kg/hour.
- Consider N-Acetylcysteine. (discuss with hepatologist). The dose is a continuous infusion at 10mg/kg/hr for at least 48-72 hours with regular serial monitoring of liver biochemical and synthetic function parameters. Small risk of anaphylaxis is present.
- Antibiotics : Combination that provides a good cover against gram negative organisms and anaerobes eg. cefotaxime and metronidazole if no specific infective agent suspected (eg. leptospira, mycoplasma)
- Antiviral : Acyclovir is recommended in neonates and small infants with Acute Liver Failure due to possibility of Herpes simplex virus infection
- Renal dysfunction
  - Possible causes : Hepato-renal syndrome, Dehydration and Low CVP/ low cardiac output. Consider haemofiltration (to discuss with Paediatric nephrologist) if supportive measures like fluid challenge, renal dose dopamine and frusemide infusion fail.

Clinical Pearls In a comatose patient:

- In the presence of sudden coma, consider intracranial bleed: request a CT Brain.
- Patients in Grade 3 or 4 coma require mechanical ventilation to maintain normal cerebral perfusion pressure.

Indication for Liver Transplantation
Paracetamol-induced disease
• Arterial pH < 7.3 (independent of the grade of encephalopathy)
OR
• Grade III or IV encephalopathy and
• Prothrombin time > 100 s and
• Serum creatinine > 3.4 mg/dL (301 µmol/l)
All other causes of fulminant hepatic failure
• Prothrombin time > 100 s (independent of grade of encephalopathy)
OR
• Any 3 of the following variables (independent of grade of encephalopathy)
• Age < 10 years or > 40 years
• Aetiology: non-A, non-B hepatitis, halothane hepatitis, idiosyncratic drug reactions
• Duration of jaundice before onset of encephalopathy > 7 days
• Prothrombin time > 50 s
• Serum bilirubin > 18 mg/dl (308 µmol/l)
Adapted from the King's College Hospital Criteria

Fluid management in liver failure		
	Normal Liver Function	Liver Failure
Volume given if no dehydration and losses are not abnormal		
Body Weight		
< 10 kg	120-150 ml/kg/day	60-80 ml/kg/day
10-20 kg	90-120 ml/kg/day	40-60 ml/kg/day
> 20 kg	50-90 ml/kg/day	30-50 ml/kg/day
Fluid type	Dextrose 4 – 5 %	Dextrose ≥ 10% (adjust according to Destrostix readings)
Potassium	1 - 3.5 mmol/kg/day	NIL WHILE ANURIC
Sodium	1.5 - 3.5 mmol/kg/day	No added sodium to existing maintenance fluid (Adjust to keep serum Na normal)
Other Fluids	Albumin 20% 5 ml/kg	Albumin 20% 5 ml/kg
For transfusion	FFP 10-20 ml/kg	FFP 10-20 ml/kg
Blood volume (ml) = No. of grams to raise Hb by x body weight in kg x F Where F = 6 for whole blood, F = 4 for packed cells		

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- 9.6. Septicaemia

- Definition: Is a suspected or proven infection plus a systemic
- inflammatory response syndrome (e.g., fever, tachycardia, tachypnea, and leukocytosis).
- Causes
- - Bacteremia: (Streptococcus pneumoniae, Haemophilus influenzae type b, Neisseria meningitidis, group A streptococcus,
- S. aureus, Salmonella)
- - Viral infection: (influenza, enteroviruses, hemorrhagic fever group, HSV, RSV, CMV, EBV)
- - Encephalitis: (arboviruses, enteroviruses, HSV)
- - Vaccine reaction (pertussis, influenza, measles)
- - Toxin-mediated reaction (toxic shock, staphylococcal scalded skin syndrome)
- Clinical evaluation
- - Assess Air way, Breathing (RR, signs of respiratory distress and pulse oximetry), Circulation (HR, BP, skin for signs of dehydration, JVP)
- - Identify SIRS (on the basis of ≥2 of the following):
- • Increased heart rate (>90/min)
- • Increased respiratory rate (>20/min) or PaCO2 <32 mm Hg

- • Increased temperature (>38°C) or decreased temperature(<36°C)
- • Increased WBC (>12,000/mm3) or decreased (<4000/mm3)
- - Identify source of infection e.g pneumonia, abdominal abscess, meningitis etc.
- - Assess organ function e.g. CNS (LOC, focal signs), renal function for urinary output
- Complications
- - Convulsions
- - Confusion or coma
- - dehydration
- - Shock
- - Cardiac failure
- - disseminated intravascular coagulation (with bleeding episodes)
- - Pneumonia
- - Septicaemic shock is an important cause of death
- Investigations
- - Identify SIRS; CBC and White-cell differential
- - Identify source of infection; blood and urine culture and
- sensitivity, sputum, CSF analysis, chest radiography and ultrasonography
- - Assess organ function;
- • Renal function: electrolytes, BUN, creatinine
- • Hepatic function: Bilirubin, AST, alkaline phosphatase
- • Coagulation: INR, PTT, platelets
- Management
- - Assess for Air way, Breathing, Circulation, dehydration and manage accordingly
- - Control the source of sepsis e.g abscess, peritonitis
- - First choice treatment:
- • I.V cefotaxime 80mg/kg/dose every 8 hours for 7 days
- Alternative
- • I.V infusion ceftriaxone 75-100mg/kg/day once over 30-60 minutes for 7 days
- Monitoring
- The child should be checked by nurses at least every 3 hours and by a doctor at least twice a day. Check for the presence of complications such as shock, reduced urine output, signs of bleeding (petechiae, purpura, bleeding from venepuncture sites), or skin ulceration.

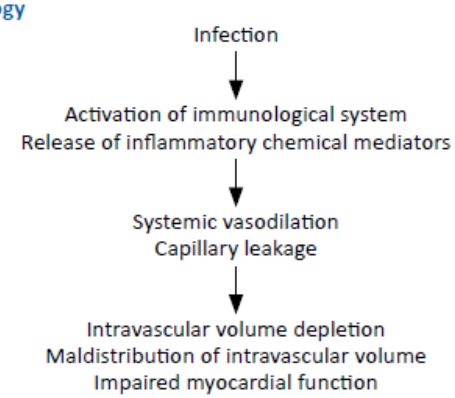
- Recommendation
- - Immunization with the conjugate H. influenzae type b and S. pneumoniae vaccines is for all infants
- Note: Use of corticosteroids in patients with sepsis has adverse effects like hyperglycemia and immunosuppression thus leading to nosocomial infection and impaired wound healing. Studies reveal that early use of short-course, high-dose corticosteroids does not improve survival in severe sepsis.

Definitions of Sepsis and Shock	
SIRS (Systemic Inflammatory Response Syndrome)	• Non-specific systemic inflammatory response to infection, trauma, burns, surgery etc. • Characterized by abnormalities in ≥ 2 of the following (one of which must be abnormal temperature or leukocyte count): • Body temperature. • Heart rate. • Respiratory function. • Peripheral leucocyte count.
Sepsis	• SIRS in the presence of or as a result of suspected or proven infection.
Severe sepsis	• Sepsis plus one of the following: • Cardiovascular organ dysfunction. • Acute respiratory distress syndrome. • Two or more other organ dysfunction.
Septic shock	• Severe sepsis with cardiovascular organ dysfunction i.e. Hypotension (systolic Blood Pressure < 5th centile for age).
Early septic shock (WARM shock)	• Compensated warm phase of shock. • Prompt response to fluids, pharmacologic treatment.
Refractory septic shock (COLD shock)	• Late decompensated phase. • Shock lasting >1 hour despite vigorous therapy necessitating vasopressor support.
Based on the International Pediatric Sepsis Consensus Conference	

Incidence

Non hospitalized immunocompetent children may develop community acquired sepsis. More commonly, hospitalized immunocompromised patients are at higher risk of developing serious healthcare associated sepsis.

Pathophysiology



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Clinical features

Sepsis, severe sepsis and septic shock are a clinical continuum.

- SEPSIS is present when 2 or more of the following features are present
  - Fever (> 38.5°C) or hypothermia, often in neonate (< 36°C)
  - Hyperventilation
  - Tachycardia
  - White blood count abnormalities: leukocytosis or leucopenia

  AND there is clinical evidence of infection.

Other constitutional symptoms such as poor feeding, diarrhea, vomiting, lethargy may be present.

- With progression to SEVERE SEPSIS, there are features of compromised end organ perfusion such as:

Features of compromised end organ perfusion	
Neurology	Altered sensorium, irritability, agitation, confusion, unresponsiveness or coma
Respiratory	Tachypnoea, increase breathing effort, apnoea / respiratory arrest, cyanosis (late sign)
Renal	Oliguria: urine output < 0.5ml/kg per hour

- When SEPTIC SHOCK sets in, look for features of *Warm* or *Cold* shock:

Features of *Warm* and *Cold* shock		
	WARM shock	COLD shock
Peripheries	Warm, flushed	Cold, clammy, cyanotic
Capillary refill	< 2 sec	> 2 sec
Pulse	Bounding	Weak, feeble
Heart rate	Tachycardia	Tachycardia or bradycardia
Blood pressure	Relatively maintained	Hypotension
Pulse pressure	Widened	Narrowed

Look out for localizing signs - most useful but not always present:

Localising Signs
Central nervous system
meningism , encephalopathy
Respiratory
localised crepitations, evidence of consolidation
Cardiovascular
changing murmurs
Gastrointestinal
focal or rebound tenderness, guarding
Bone and soft tissue
focal erythema, tenderness and oedema
Head and neck
cervical lymphadenopathy, sinus tenderness,
inflamed tympanic membrane, stridor,
exudative pharyngotonsillitis
Skin
pustular lesions

Complications

Multiorgan Failure:

- Acute respiratory distress syndrome.
- Acute renal failure.
- Disseminated intravascular coagulopathy.
- Central nervous system dysfunction.
- Hepatic failure.

-

Investigations	
Septic work - up	**Monitoring severity and progress**
• Blood C&S	• Full blood count
• Urine C&S	• Renal profile
Where appropriate	• Electrolytes, calcium, magnesium
• CSF C&S	• Blood sugar
• Tracheal aspirate C&S	• Blood gases
• Pus / exudate C&S	• +/- lactate levels
• Fungal cultures	• Coagulation profile
• Serology, viral studies	• Liver function test
• Imaging studies	
- Chest X-ray, ultrasound, CT scan	
Supporting evidence of infection:	
Full blood count	
Leukocytosis or leukopenia	
Peripheral blood film	
Increase in immature neutrophil count	
C-reactive protein	
Elevated c-reactive protein levels	
Abbreviation. C&S, Culture and Sensitivity	

Management

- Initial resuscitation - ABC
  - Secure airway, Support breathing, Restore circulation

Caution: the use of sedation in septic or hypotensive children may result in crash of blood pressure. If sedation is required, use low dose IV Midazolam or Ketamine, volume infusion should be continued and inotropes should be initiated, if time permits.

- Fluid therapy
  - Aggressive fluid resuscitation with crystalloids or colloids at 20 mls/kg as rapid IV push over 5-10 mins. Can be repeated up to 60 mls/kg or more.
  - Correct hypoglycaemia and hypocalcaemia.
- Inotropic Support
  - If fluid refractory shock*, establish central venous access
    - Start inotropes: IV Dopamine 5 - 15 µg/kg min *or*
    - IV Dobutamine 5 - 15 µg/kg/min
  - For fluid refractory and dopamine/dobutamine refractory shock with
    - Warm shock : titrate IV Noradrenaline 0.05 – 2.0 µg/kg /min
    - Cold shock : titrate IV Adrenaline 0.05 – 2.0 µg/kg /min
  - The aim of titration of inotropes include normal clinical endpoints and where available, SpO_2 >70%.
  - Inotropes should be infused via a central line (whenever possible) or a large bore peripheral canula.
  - Use dedicated line or lumen. Avoid concurrent use for other IV fluids, medication.
  - Fluids and inotropes to be titrated to optimal vital signs, urine output and conscious level.

**hypotension, abnormal capillary refill or extremity coolness*

- Antimicrobial therapy
  - IV antibiotics should be administered immediately after appropriate cultures are taken. Start empirical, broad spectrum to cover all likely pathogens, considering:
    - Risk factors of patient and underlying illness.
    - Local organism prevalence and sensitivity patterns.
    - Protocols of the institution.
  - Antibiotic regime to be modified accordingly once C&S results are back.
  - Source control:
    - Evaluate patient to identify focus of infection.
    - Drainage, debridement or removal of infected devices to help control infection.

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- Respiratory Support
  - Use PEEP and FIO_2 to keep $SaO_2 > 90\%$, $PaO_2 > 80$ mmHg

Caution: use sufficient PEEP to ensure alveolar recruitment in cases of sepsis with acute lung injury. Too high PEEP can result in raised intrathoracic pressure which can compromise venous return and worsen hypotension.

- Supportive Therapy
  - Packed cells transfusion if Hb <10g/L.
  - Platelet concentrate transfusion if platelet count < 20 000/mm^3.
  - If overt clinical bleeding, correct coagulopathy or DIVC.
  - Bicarbonate therapy: give bicarbonate only in refractory metabolic acidosis, if pH < 7.1 (ensure adequate tissue perfusion and ventilation to clear by-product CO_2).
  - Aim to maintain normal electrolytes and blood sugar.
- Monitoring
  - Frequent serial re-evaluation is essential to guide therapy and gauge response, as below:

Monitoring in Children with Sepsis
Clinical
• *Vital signs*
• Heart rate via cardiac monitor
• Capillary return
• Skin temperature
• Pulse volume
• Blood pressure
• Non invasive
• Invasive - ideal if available
• SpO_2 via pulse oximeter
• Central venous pressure (CVP)
Urine output via continuous bladder drainage
Head chart (GCS)
Laboratory
See previous Table on Investigations

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- 9.7. Salmonella Infections (Typhoid Fever)
- Definition: is a systemic infection with the bacterium Salmonella
- enterica serotype typhi.

- Cause
- - Bacteria (Salmonella typhi)
- Signs and Symptoms
- - Fever and malaise
- - dull frontal headache
- - Poorly localized abdominal discomfort
- - Anorexia, nausea and diarrhea or constipation
- - A coated tongue, tender abdomen, hepatomegaly, and
- splenomegaly are common
- - Febrile convulsions
- - Jaundice may occur
- Note: There is no thyphoid fever without fever or hypothermia in infants!!!
- Diagnosis
- - On examination, key diagnostic features of typhoid are:
- • Fever with no obvious focus of infection
- • No stiff neck or other specific signs of meningitis, or a
- lumbar puncture for meningitis is negative (note: children
- with typhoid can occasionally have a stiff neck)
- • Signs of systemic upset, e.g. inability to drink or breastfeed,
- convulsions, lethargy, disorientation/confusion, or
- vomiting
- • Rose spots on the abdominal wall (in light-skinned
- children)
- • Hepatosplenomegaly, tense and distended abdomen
- Typhoid fever can atypically be present in young infants
- as an acute febrile illness with shock and hypothermia. In
- areas where typhus is common, it may be very difficult to
- distinguish between typhoid fever and typhus by clinical
- examination alone. The differential diagnosis is broad and
- includes malaria, amebiasis, dengue fever, leishmaniasis, and other causes of bacterial gastroenteritis.
- Diseases
- Complications
- - GIT: gastrointestinal bleeding, intestinal perforation, abdominal
- mass due to abscess formation
- - CVS: Asymptomatic electrocardiographic changes, myocarditis, shock
- - CNS: Encephalopathy, delirium, psychotic behaviour, meningitis, impairment of coordination

- - Hematologic: Anemia, disseminated intravascular coagulation
- - Respiratory: Bronchitis, pneumonia (salmonella enterica
- serotype typhi, streptococcus pneumoniae)
- - Cardiovascular (myocardite)
- - Others: Focal abscess, pharyngitis, relapse and chronic carriage
- - Chronic carriers frequently have high serum antibody titers
- against the Vi antigen, which is a clinically useful test for rapid identification of such patients
- Investigations
- - FBC (may show leucocytosis more common in children or leucopenia, thrombocytopenia, severe anaemia follows intestinal
- bleeding)
- - Blood culture (Gold standard) will isolate the bacteria during the first 2 weeks of illness
- - Stool culture will isolate the bacteria during the later period of illness
- - Plain x-rays of abdomen in erect position will show gas under the diaphragm if there is gut perforation
- Note: Bone marrow cultures may be positive in as many as 50% of patients after as many as five days of antibiotics.
- Serology — Serologic tests such as the Widal test are of limited clinical utility in endemic areas because positive results may represent previous
- infection. Positive serology only, shall never be a base for treatment of typhoid fever (several clinicians in Rwanda are still using this)
- Management
- Pharmacological
- • Paracetamol to reduce fever
- • Rectal Diazepam if there are convulsions and blood
- transfusion in case of severe bleeding
- • ciprofloxacin 10mg/kg (max400mg) every 12 hours
- ciprofloxacin 15mg/kg (max500mg) orally every 12 hours
- for7-10 days
- • ceftriaxone 50 mg/kg every 12 hours IV for 7-14 days
- Or
- • cefotaxime 50 mg/kg IV every 6 hours for 7-14days
- Follow up review: check for the following
- • Efficacy of treatment: fever
- • Perforation (abdominal pain, tenderness, transit)

- • Myocarditis (cardiac frequency, cardiac auscultation)

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